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L. G. Wong, *Shaklee U.S., Inc., San Francisco, California*

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Ames Research Center
Moffett Field, CA 94035-1000

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Ames Research Center

Summary

To test the hypothesis that drink composition is more important than drink osmolality (Osm) for maintaining and increasing plasma volume (PV) at rest and during exercise, six men (22–39 yr, 76.84 ± 16.19 kg, 2.99 ± 0.45 L/min $\dot{V}O_2$ peak) each underwent six treatments while sitting for 90 min ($\dot{V}O_2$ = 0.39 L/min) and then performed upright ergometer exercise for 70 min ($\dot{V}O_2$ = 2.08 ± 0.33 l/min, 70% ± 7% $\dot{V}O_2$ peak). Drink formulations (10 ml/kg body weight, $\overline{X}$ = 768 ml) for the sitting period were: P1 (55 mEq Na^+, 365 mOsm/kg H_2O), P2 (97.1 mEq Na^+, 791 mOsm/kg), P2G (113 mEq Na^+, 80 ml glycerol, 1,382 mOsm/kg), HyperAde (HA) (164 mEq Na^+, 253 mOsm/kg), and 01 and 02 (no drinking). The exercise drink (10 ml/kg, 768 ml) was P1 for all treatments except 02. Plasma volume at rest increased ($p < 0.05$) by 4.7% with P1 and by 7.9% with HA. Percent change in PV during exercise was +1% to +3% (NS) with HA; –6% to 0% (NS) with P1, P2, P2G, and 01; and –8% to –5% ($p < 0.05$) with 02. HyperAde, with the lowest osmolality (253 mOsm/kg), maintained PV at rest and during exercise, whereas the other drinks with lower Na^+ and higher osmolality (365 to 1,382 mOsm/kg) did not. But Performance 1 also increased PV at rest. Thus, drink composition may be more important than drink osmolality for increasing plasma volume at rest and for maintaining it during exercise.

Introduction

The mechanism of muscular fatigue caused by physical work and exercise (high metabolism) is not clear, but it involves disturbance of muscle surface membrane excitation–contraction coupling as a result of changes in sarcoplasmic reticulum Ca^{2+} release, cell H^+ and inorganic P responses, and carbohydrate metabolism (Fitts 1994). Low-metabolism fatigue in people at rest involves both psychological and physiological factors (Bartlett 1953, Booth 1991), probably in various proportions. One common factor appears to be the concentration and distribution of water and electrolytes within muscle cells and other body fluid compartments (vascular, interstitial, and cellular). The vascular fluid volume, composed of plasma and red blood cells, is a primary regulator of cardiovascular function; reduction of this volume (hypovolemia) and total body water (hypohydration) adversely affects exercise performance (Greenleaf 1973). In addition, plasma volume and ionic–osmotic constituent concentration of plasma and cells are also regulatory factors for body thermoregulation, which is often compromised with exercise-induced hypovolemia and hypohydration (Greenleaf and Castle 1971, Greenleaf 1979, Kozlowski et al. 1980).

Rehydration of dehydrated people is relatively easy with appropriate food (osmols), fluid, and a restful environment. But ad libitum fluid intake under stressful conditions, e.g., heat, exercise, or prior dehydration, results in involuntary dehydration (Greenleaf 1991, 1992; Rothstein et al. 1947) defined as the delay in full fluid replacement (euhydration) during and following loss of body fluid. Stress caused by doing mental arithmetic can also cause hypovolemia (S. Patterson, personal communication). Thus, people subjected to acute or chronic stress may be somewhat "dehydrated" as well as fatigued.

Research on body fluid distribution and rehydration fluid composition, stimulated by demands on troops during World War II (Adolph 1947, Pitts et al. 1944), has continued with increasing intensity for military personnel (Marriott 1994) with application for recreational exercisers and competitive athletes (Murray 1987). Many current rehydration formulations are more concentrated (hypertonic–hyperosmotic) than the normal plasma osmolality (285 mOsm/kg H_2O) with more of the drink osmols contributed by carbohydrate than by ionized solute (Murray 1987). Optimal fluid composition for rapid gastric emptying and transfer through the gastrointestinal system appears to be 20–30 mEq/L sodium, 5–10 mEq/L potassium (with chloride as the only anion), and 0.9%–10% carbohydrate, preferably glucose (Gisolfi 1991). Measurement of gastric and gastrointestinal emptying of fluid does not necessarily reflect change in plasma or interstitial fluid volumes. There have been few

*Shaklee U.S., Inc., San Francisco, California.

studies on the efficacy of various drink formulations for increasing body fluid compartment volumes, especially plasma volume (PV) in rested hydrated subjects (Luetkemeier and Thomas 1994; Maughan and Noakes 1991, Murray 1987).

Recent findings in our laboratory indicated that fluid formulations containing greater concentration of ionized solute (Performance 1 and HyperAde) up to 164 mEq/L Na^+ induce significantly ($p < 0.05$) greater levels of hypervolemia in resting, moderately dehydrated men, and are also better than water for attenuating the hypovolemia during supine, submaximal, leg ergometer exercise (Greenleaf et al. 1992). The present study was designed from these preliminary findings to determine the effect of intermittent ingestion of two previously tested and two newly formulated hypertonic solutions containing various osmotic and carbohydrate concentrations on plasma volume during rest followed by upright submaximal ergometer exercise. To test the physiological effect of the hyperhydration, thermoregulatory parameters were measured.

The authors thank the subjects for their dedication and cooperation. This study was supported by Shaklee Grant JSRA-7 and NASA Grants 199-18-12-07 and NAG8-227.

Methods

Subjects

Six men, aged 22–39 yr, (table 1) gave written informed consent for this study which was approved by the Ames Research Center Human Research Experiments Review Board and the San Francisco State University Human Subjects' Committee. The men passed a comprehensive medical examination which included their medical history, urine and blood analyses, and a treadmill exercise test. All were nonsmokers and none took nonprescribed drugs.

Procedure

Six treatments for each subject were conducted semi-randomly at weekly intervals. The experimental protocol consisted of intermittent drinking during 90 min of sitting rest, 15 min to move to the cycle ergometer and to readjust sensors, intermittent drinking during 70 min of upright submaximal (70% ± SD 7% of peak oxygen uptake) leg exercise, followed by 10 min of sitting recovery (fig. 1).

The subjects arrived at the Laboratory for Human Environmental Physiology at 0700 hr and ate a standardized carbohydrate breakfast: 220 ml of reconstituted frozen orange juice and two toasted English muffins with jelly. After breakfast they urinated and inserted a rectal thermistor 16 cm. Dressed in shorts (weighed dry), they were weighed (±5 g) on a digital scale (model 5780, National Controls, Inc., San Carlos, California). The men then sat in a chair for 90 min while skin probes and sensors (EKG, laser-Doppler, temperature, and sweat capsules) were attached and a forearm venous catheter (Quik-Cath, Travenol Laboratories, Inc., Deerfield, Illinois) was inserted. Body weight was measured and additional urine samples were collected after the rest and exercise periods (fig. 1).

Drinks and Drinking

Each subject drank one of four fluid formulations (table 2), divided into seven portions, during the rest and exercise periods (fig. 1). The drinks were designated P1 (Performance 1), P2 (2 × Performance 1 concentration), P2G (P2 + 80 ml glycerol), HA (HyperAde), or 0 (no drinking). Performance 1 is a commercial product of Shaklee U.S., Inc., San Francisco, California; HyperAde was also packaged by Shaklee. All drinks, in powder form, were mixed just prior to testing. The high salt content of HA, as well as the very sweet taste of P2G, were apparent to the subjects. Drink volume was 10 ml/kg body weight for both resting and exercise phases (table 3). Glycerol was used for its water retaining properties. Performance 1 was consumed during exercise with five treatments, and no drinking was done during the sixth. Thus, for the six treatments, drink designations for rest/exercise, respectively, were: P1/P1, P2/P1, P2G/P1, HA/P1, O/P1, and 0/0.

Physiological Measurements

After three familiarization sessions, the peak oxygen uptake ($\dot{V}O_2$ peak, table 1) was measured with the subjects in the upright sitting position on a model 846 cycle ergometer (Quinton Instruments Co., Seattle, Washington). The respiratory measurement system utilized a low-resistance, low-dead-space Rudolph Valve (model 2700, Hans Rudolph, Inc., Kansas City, Missouri), a Tissot-tank calibrated electronic spirometer (model S-301 Pneumoscan, K.L. Engineering Co., Sylmar, California), and a 3-L mixing chamber from which expired gas was sampled at 0.5 L/min, drawn through and dried by anhydrous calcium sulfate (N.A. Hammond Drierite Co., Xenia, Ohio) and routed to oxygen and carbon dioxide analyzers (Applied Electrochemistry models S-3AI and CD-3A, respectively; Ametek, Thermox Instruments Division, Pittsburg, Pennsylvania). The analyzers were calibrated with standardized gases (Lloyd-Haldane apparatus).

Analog data were processed on-line with an analog-to-digital converter (VISTA system IBM model 17002, Vacumed, Ventura, California) and transmitted to an IBM (model AT) computer; output metabolic data were printed each 15 sec. Peak data were the mean of the final four 15-sec values. The submaximal exercise load corresponded to an oxygen uptake of 70% ± SD 7% of the measured $\dot{V}O_2$ peak (table 4).

Skin blood velocity was measured on the left temple and left anterior-medial thigh with a laser-Doppler system (model BPM 403A, LaserFlo Blood Perfusion Monitor, TSI Inc., St. Paul, Minnesota). Heart rate was determined with a cardiotachometer (model 78203C, Hewlett-Packard, Waltham, Massachusetts) via three skin electrodes (Silvon No. 01-3630 Ag/AgCl, NDM, Dayton, Ohio), two located on the anterior shoulders and the third over the fifth intercostal space.

Local sweat rate was measured via capsules (Spaul 1983) on the left arm, forearm, and anterior thigh with resistance hygrometry sensors (model 2300 H1T21, Thunder Scientific Corp., Albuquerque, New Mexico); room air was the reference. The sweat capsules were located adjacent to skin-temperature thermistors. The sensors were calibrated with solutions of standard humidity: 33% with $MgCl_2$, 52% with $MgNO_3$, and 96.5% with K_2SO_4, and room air (43.5%) was measured with a psychrometer. Regression equations for measured versus actual humidities were: thigh ($Y = 1.10\,X - 0.78$, $r = 0.99$), forearm ($Y = 1.03\,X - 4.97$, $r = 0.98$), and arm ($Y = 1.15\,X = -13.40$, $r = 0.99$). Sensor and room air relative humidities were recorded with a DigiTech Datalogger (model 1100, United Systems Corp., Dayton, Ohio). Sweat rate ($\dot{M}_{sw}$) at the three sites was calculated as follows: $\dot{M}_{sw} = [\dot{V}_a(W_{out} - W_{in})]/[(A_{sw})(SVA)]$ where $\dot{M}_{sw}$ = sweat rate ($g \cdot cm^{-2} \cdot hr^{-1}$), $\dot{V}_a$ = volume flow rate (L/min), W_{out}, = absolute humidity ratio leaving capsule (lb H_2O/lb dry air), W_{in} = absolute humidity ratio of control (lb H_2O/lb dry air), A_{sw} = area of collection capsule (3.14 cm^2), and SVA = specific volume of air (L/g dry air).

Body water balance (gross sweat rate) was calculated: balance = (weight loss – blood + urine loss + drink volume) – (CO_2 out – O_2 in).

Rectal and skin temperatures were measured with series 400 thermistors (Yellow Springs Instrument Co., Yellow Springs, Ohio). Skin thermistors, attached with holders that permitted free movement of air (Greenleaf and Williams 1976), were located at six sites: arm, forearm, thigh, calf, chest, and back. A Squirrel meter/ logger (Grant model 1200, Science/Electronics Inc., Miamisburg, Ohio) was used for processing sensor signals. Mean skin temperature ($\overline{T}sk$) (Greenleaf and Castle 1972, Hardy and Dubois 1938) was calculated: $\overline{T}sk$ = 0.06 (Tarm) +0.13 (Tforearm) +0.21 (Tthigh) +0.21 (Tcalf) +0.19 (Tchest) +0.20 (Tback). Mean room dry-bulb temperature was 21.8°C ± SD 0.3°C, and relative humidity was 50% ± 2% (table 5). A fan increased airflow over the subject during rest (23 ± SD 4 ft/min) and exercise (53 ± 4 ft/min).

Blood Measurements

Blood samples (15 ml each (20 ml each at –25 and –35 min), 115 ml/experiment) were withdrawn through an 18-gauge catheter (Quik-Cath, Baxter Healthcare Corp., Deerfield, Illinois) inserted into the right antecubital vein. Blood samples were divided in four Vacutainer[R] tubes: tube 1 = 2 ml for hemoglobin (Hb) and hematocrit (Hct); tube 2 = 3 ml for glucose; tube 3 = 10 ml for sodium, potassium, osmolality, RBC, WBC, platelets, and glycerol; and tube 4 = 5 ml for Evans blue (plasma volume) analysis. Hemoglobin and Hct were measured immediately (manually). Hemoglobin was measured (cyanomethemoglobin method) with the Coulter Diluter II and Hemoglobinometer (Coulter Electronics, Hialeah, Florida). Blood for Hct was drawn into four capillary tubes, centrifuged for 10 min at 11,500 rpm (centrifuge model MB, International Equipment Co., Needham Heights, Massachusetts) and read with a modified micro-capillary tube reader (model CR, International Equipment Co.). Hemoglobin and Hct were also calculated automatically with a Coulter model STKS analyzer. Plasma was frozen (–20°C) for subsequent analysis.

Plasma sodium, potassium, glucose, citrate, and glycerol concentrations were measured with a Cobas Mira S analyzer (Roche Diagnostic Systems, Inc., Branchburg, New Jersey): sodium (glass membrane) and potassium (PVC valinomycin membrane) with ion-selective electrodes; glucose with hexokinase–NAD reactions and NADH read at 340 nm; glycerol with glycerolkinase–glycerophosphate oxidase–peroxidase reactions with the quinoneimine complex read at 490–550 nm; and citrate with citrate lyase for NADH to NAD^+ at 340 nm. Plasma osmolality was measured by freezing-point depression (model 3DII, Advanced Instruments Digimatic Osmometer, Needham Heights, Massachusetts).

Plasma volume was measured on frozen plasma with the Evans blue dye (T-1824, New World Trading Corp., DeBary, Florida) dilution technique from one 10-min post-dye-injection blood sample (Campbell et al. 1958, Greenleaf et al. 1979). Freezing does not change T-1824 concentration over time. Plasma was eluted through machine-packed chromatographic columns (model PD-10, Sephadex G-25M, Pharmacia LKB, Uppsala, Sweden) and the elutriate was read at 615 mμ. Plasma volume was calculated: $PV = (V \cdot D \cdot St \cdot v)/(T \cdot 1.03)$ where V = volume T-1824 injected, D = dilution of standard, St = standard absorbance, v = volume of sample extracted, T = test sample absorbance (subtract plasma blank), and 1.03 = correction factor for slow dye uptake by tissues. Percent change in plasma volume was calculated using the Hb–Hct transformation equation (Greenleaf et al. 1979).

Data from the new Sephadex column were compared with data from the standard manually packed column (Greenleaf 1979b). The optical density of 0.2 ml T-1824/10 ml acetone standard was measured (0.130); then 0.2 ml T-1824 was mixed with Teepol-phosphate and eluted through nine manually packed chromatographic columns and nine Sephadex columns. Mean (±SD and ±SE) optical density for the manual and Sephadex columns was 0.1103 (±0.0041 and ±0.0014) and 0.0949 (±0.0026 and ±0.0008), respectively ($\Delta \bar{X} = 14.0\%$, $p < 0.0001$). Thus, optical density from the Sephadex column was lower and variability of the elutriate was about half that of the manually packed column.

Mean corpuscular volume (MCV, μ^3) = 10 (Hct · 0.96)/ (RBC in $10^6/mm^3$).

Hematocrit and hemoglobin concentration were determined manually (as indicated above) and with calculated values from the Coulter counter (fig. 2). The calculated Hb values were lower and the Hct values were higher than their respective manual values which were used for the plasma and blood volume determinations.

Urine Measurements

The volume of urine, collected at the end of rest (–15 min) and after exercise (+10 min of recovery), was timed and measured in a graduated cylinder. Urinary excretion rate ($\dot{V}$) was expressed in mL/min. Urinary sodium (U_{Na}), potassium (U_K), and osmotic (U_{osm}) concentrations were determined by the same methods used for the respective plasma variables. Other urine functions were calculated: osmotic clearance (C_{osm}) was urine osmotic excretion ($U_{osm}\dot{V}$) divided by plasma osmolality (P_{osm}) averaged over the urine collection period; free water clearance (C_{H_2O}) was $\dot{V} - C_{osm}$; and fractional ionic excretion was $U_{Na}\dot{V}$ and $U_K\dot{V}$.

Statistical Analysis

The data were analyzed, as a first approximation, by Student's t-test for dependent variables. The null hypothesis was rejected when $p < 0.05$. Nonsignificant differences were denoted by NS or trend or tendency.

Results and Discussion

Blood Data

Plasma and mean corpuscular volume– Percent change in plasma volume from –105 min (upper panel), and from –105 min (rest) and from 0 min (exercise) (lower panel), are presented in figure 3. At the end of the rest phase (–15 min) the greater ($p < 0.05$) increase in PV occurred with the HAP1 (by 7.9%) and P1P1 (by 4.7%) treatments; the lesser increase was with the 00 (by 1.7%) and 0P1 (by 1.0%) treatments. Change from sitting upright in a chair with the thighs horizontal, to sitting upright on the cycle with thighs positioned at a more downward angle (position change) resulted in decreasing trends in PV at time zero with all treatments which resulted from the increased hydrostatic pressure in the lower extremities. Percent change in PV with 0P1 at time zero was similar to that of 00, so the two no-drinking treatments responded similarly. During exercise, HAP1 maintained the highest PV, followed by P1P1, 0P1, P2GP1, P2P1, and 00 in decreasing order (fig. 3, upper panel). Thus, drinking P1 during exercise by dehydrated subjects can increase PV to the hydrated-control level. Reduction in PV by 4% to 9% occurred with all treatments at 10 min of exercise, with essentially similar rates of recovery regardless of whether or not fluid was consumed (fig. 3, lower panel). The 00 response was similar to the P2GP1 response. Thus, the rate of PV restitution during exercise appeared to be independent not only of drink composition, but also of whether or not fluid was consumed.

Mean corpuscular volumes (fig. 4) were not different from each other or over time during rest or exercise, indicating that there was no appreciable exchange of vascular fluid into or from red blood cells.

Osmolality– Plasma osmotic concentration was within the upper half of the normal range (277–297 mOsm/kg H_2O) and varied between 288 and 293 mOsm/kg H_2O in the rest phase (fig. 5, upper panel). In both nondrinking treatments (0P1 and 00), plasma osmolality remained constant during the first hour of rest. Osmolality varied by ±2 mOsm/kg by the end of rest; P1P1 and P2GP1 exhibited positive changes and HAP2 and 0P1 exhibited negative changes (fig. 5, middle panel). All osmotic responses were within the normal variability. Plasma osmolality increased during exercise with all treatments, especially 0P1 (with drinking P1) and 00 (with no drinking). Intake of P1 had no apparent effect on change in osmolality. Drinks P2GP1 and HAP1 had the lower osmotic concentration at the end of exercise (fig. 5, upper panel) which accompanied the greater increase in plasma volume. As expected, treatment 00 exhibited the greatest increase in osmolality by the end of exercise; HAP1 had the least increase (fig. 5, middle panel). Also, HAP1, with the highest ionic osmolality, had the greatest increase in plasma osmotic content; osmotic content of the remaining treatments returned to normal by the end of exercise (fig. 5, lower panel). The acute decrease in plasma osmotic content at the beginning of exercise accompanied, and possibly induced, the shift of plasma from the vascular space.

Sodium – Plasma sodium concentration generally followed comparable osmotic concentration, especially when respective percent change in content was compared (figs. 5 and 6, lower panels). Because sodium and accompanying anions account for a large part of plasma osmolality (plasma sodium and osmotic concentrations $r = 0.93$), the osmotic contribution of carbohydrates was minimal.

Potassium – Plasma potassium was within the normal range at rest (fig. 7, upper panel) and, unlike sodium, both potassium concentration and content exhibited immediate increase with the onset of exercise (fig. 7, lower panel). The potassium content in the drinks did not appear to influence the concentration or content responses at rest or during exercise. At 70 min of exercise the greater percent change in content occurred in HAP1, 0P1, and P2P1 (containing potassium), and the smallest change occurred in P1P1 (also containing potassium), with 00 (containing no potassium) in the middle (fig. 7, lower panel). Thus potassium, the major intracellular ion, did not accompany the shift of sodium and water from the vascular space at the beginning of exercise.

Glucose – Plasma glucose was elevated above the normal range of 64–115 mg/dL at the beginning of the rest period, probably a result of the high-carbohydrate breakfast (fig. 8, upper panel). Glucose concentration decreased with all treatments during rest and position change, with a greater decrease for those with no carbohydrate (HAP1, 00, 0P1). With the exception of HAP1, glucose concentration and content decreased immediately with the onset of exercise (similar to osmolality and sodium), and then increased as exercise continued (fig. 8, lower panel). Glucose concentration for treatments 0P1 and 00 were similar at time zero, but by the end of exercise that of 0P1 increased the most (to 110 mg/dL) and that of 00 increased the least (to 85 mg/dL) by 70 min (fig. 8, upper panel). Similar results were evident with changes in glucose concentration and content. Thus, consumption of glucose during exercise increased both plasma glucose concentration and content.

Glycerol – Only one drink (P2G) contained appreciable (80 mL) glycerol. Plasma glycerol increased to 168 ± 33 mg/dL at zero min of rest, remained at that level during the first 30 min of exercise, and then decreased to 116 ± 18 mg/dL at 70 min (fig. 9, upper and lower panels). Apparently there was some glycerol metabolism: the change in glycerol content decreased from 3,462% ± 1,430% at zero min to 2,208% ± 768% at 70 min of moderately heavy ergometer exercise.

Citrate – Mean resting plasma citrate varied from 1.7 ± 0.2 to 2.2 ± 0.3 mg/dL, within the normal range of 1.7 – 3.0 mg/dL (fig. 10, upper panel; appendix 2). Citrate was present in all drinks: 3.87 g/2 L in P1, 7.74 g/2 L in P2 and P2G, and 15.44 g/2 L in HA (table 2). Plasma citrate increased by 0.5 pg/mL (P2G) to 1.7 pg/mL (HA), and remained essentially constant with 0P1 and 00 at zero min (fig. 10, lower panel). In spite of the fact that drink P1 was consumed during exercise with all treatments except 00, citrate concentration in the four rest citrated drinks converged at about 0.75 mg/dL at rest, with a pronounced decrease in citrate with HA as consumption changed from 15.44 g/2 L at rest to 3.87 g/2 L during exercise. Reducing citrate consumption by 50% from rest to exercise did not appreciably alter the change in citrate content in the P1P1, P2P1, and P2GP1 treatments (fig. 10, lower panel).

Urine Data

Excretion rate and electrolyte–osmotic concentration – Urine excretion rate ($\dot{V}$) at rest varied from 1.2 ± 0.3 mL/min (0P1) to 3.2 ± 1.2 mL/min (P2GP1), with a mean level ($N = 6$) of 2.3 ± 0.3 mL/min (fig. 11, solid line). Normal resting $\dot{V}$ is 1.0 mL/min. Excretion rate during exercise varied from 0.8 ± 0.3 mL/min (0P1 and 00) to 3.2 ± 0.8 mL/min (HAP1), with a mean rate ($N = 6$) of 1.8 ± 0.4 mL/min (fig. 11, dashed line) which was not significantly lower than the rest mean rate. Exercise $\dot{V}$ was depressed similarly with P2P1, 0P1, and 00, but not with P2GP1 or HAP1 with their higher osmotic concentrations.

In general, urine sodium, potassium, and osmotic concentrations were lower with P1P1 and P2P1, and higher with HAP1, 0P1, and 00 treatments (table 6). The former reflected the lower drink osmolality, while the latter resulted from the greater ionic content of HAP1 (in spite of its lower osmolality); the urine response to dehydration was similar to that following high salt consumption. The somewhat elevated urine potassium concentration during exercise over that at rest resulted from increased muscle activity.

Sodium excretion – Mean (±SE) sodium excretion for the six treatments was 168 ± 19 μEq/min ($p < 0.05$) during exercise (–15 to +10 min) (fig. 12, upper panel). The large increase in $U_{Na} \cdot \dot{V}$ during rest and exercise with

HAP1 was due to its high sodium concentration (164 mEq/L).

Potassium excretion– There was no significant difference between mean $U_K \cdot \dot{V}$ at rest (58 ± 8 μEq/min) and during exercise of 75 ± 20 μEq/min (fig. 12, lower panel). The large increase in potassium excretion with HAP1 during exercise probably accompanied the fluid shift from muscle cells to the interstitial and vascular spaces.

Osmotic clearance– There was no significant difference between mean $U_{Osm} \cdot \dot{V}/P_{Osm}$ at rest (3.0 ± 0.2 mL/min) and during exercise (2.4 ± 0.4 mL/min) (fig. 13, upper panel). The somewhat increased osmotic clearance with HAP1 during exercise reflected the increased concomitant excretion of sodium and potassium.

Free water clearance– There was no significant difference between mean free water clearance (C_{H_2O}) at rest (–0.74 ± 0.23 mL/min) and during exercise (–0.60 ± 0.24 mL/min) (fig. 13, lower panel). Treatments with higher ionic content (HAP1) and dehydration (0P1 and 00) have the least C_{H_2O}, suggesting greater water retention.

Physiological Data

Heart rate– Mean heart rate varied from 71 ± 6 to 87 ± 8 beats/min during the rest phase to 149 ± 9 to 160 ± 8 beats/min at 70 min of exercise (fig. 14, upper panel). The increase in heart rate during exercise was lowest (61 ± 10 beats/min) with P1P1, and greatest (74 ± 10 beats/min) with HAP1 (fig. 14, lower panel). Dehydration (00) did not result in the characteristic elevated heart rate at rest or during exercise.

Rectal and mean skin temperatures– Mean (±SE) rectal temperature (Tre) was stable with each treatment at rest; it varied from 36.6 ± 0.2°C with P2GP1 to 37.2 ± 0.1°C with 0P1 (fig. 15, upper panel). The range and variability of Tre decreased by time zero. Equilibrium levels of Tre at min 70 of exercise varied from 37.98 ± 0.10°C with P1P1 to 38.29 ± 0.17°C with 0P1. Mean change in Tre during exercise (fig. 15, lower panel) did not exhibit the expected response where the 0P1 and 00 changes in Tre should have been the greatest. In fact, P2GP1 showed the greatest increase (1.41 ± 0.13°C); followed by P2P1 (1.34 ± 0.17°C), 00 (1.33 ± 0.14°C), HAP1 (1.31 ± 0.14°C), 0P1 (1.25 ± 0.15°C), and P1P1 (1.14 ± 0.08°C). Thus it appears that glycerol ingestion tends to elevate Tre whereas P1 tends to attenuate the increase in Tre.

Absolute average mean skin temperatures $(\overline{T}sk)$ (fig. 16, upper panel) and the change in $\overline{T}sk$ (fig. 16, lower panel) were not significantly different between the six treatments. Treatment 00 $\overline{T}sk$ was nearest zero, while treatment 0P1 tended to have the greater decrease (fig. 16, lower panel). Lower $\overline{T}sk$ suggests greater sweating and evaporative heat loss.

Forearm and thigh sweat rates– Mean (±SE) rest (time zero) forearm sweat rate varied from 0.02 ± 0.02 mg/min · cm^2 (0P1) to 0.16 ± 0.09 mg/min · cm^2 with P2GP1 (fig. 17, upper panel). Sweat rate was unchanged for the first 10 min of exercise, when all rates began to rise to reach 0.22 ± 0.09 mg/min · cm^2 (00) to 0.49 ± 0.11 mg/min · cm^2 (0P1). Change in forearm sweat rate responded similarly where 00 increased least (as expected) by 0.17 ± 0.07 mg/min · cm^2; and 0P1 increased most by 0.47 ± 0.10 mg/min · cm^2 (fig. 17, lower panel), suggesting enhanced sweating when dehydration at rest precedes drinking during exercise.

Thigh sweat rate at rest (time zero) was slightly higher than forearm sweat rate (fig. 18, upper panel): it varied from 0.05 ± 0.02 mg/min · cm^2 (0P1) to 0.20 ± 0.03 mg/min · cm^2 (P2GP1). Rates began to increase after 5 min of exercise to reach 0.46 ± 0.05 mg/min · cm^2 (HA) to 0.060 ± 0.07 mg/min · cm^2 (P2GP1). Change in thigh sweat rate followed a similar pattern; HAP1 increased least by 0.34 ± 0.04 mg/min · cm^2, and 0P1 increased most by 0.46 ± 0.08 mg/min · cm^2, similar to the forearm sweating response. However, the change in 00 thigh rate was also increased similar to that of the 0P1 rate, unlike the change in forearm sweating where 00 had the most attenuated rate.

Body water balance and sweat rate– Mean (±SE) body water balance for the six treatments was 42 ± 76 mL at rest, and –650 ± 81 mL ($p < 0.01$) during exercise (fig. 19). Treatments P1P1 and P2P1 resulted in greater positive balance and HAP1 had greater negative balance at rest, indicating increased sweating, whereas P2P1 had the greatest negative balance and P2GP1 and HAP1 the lesser negative balances during exercise, indicating reduced sweating (table 7). Treatments 0P1 and 00 had virtually similar unchanged rest balances and negative exercise balances, indicating that the latter were not affected by consuming P1 (fig. 19).

Temple and thigh skin blood velocity– Mean (±SE) temple skin blood velocity (from an inactive area of the body) was constant at rest, and varied from 0.35 ± 0.05 to 0.62 ± 0.07 Hz · 10^2 among the subjects (fig. 20, upper panel). All temple velocities increased after 5 min of exercise and, after about 55 min, reached equilibrium between 0.77 ± 0.14 and 0.98 ± 0.15 Hz · 10^2. Treatment HAP1 had the lowest (0.26 ± 0.11 Hz · 10^2) and 0P1 the highest (0.50 ± 0.13 Hz · 10^2) increase in velocity at 70 min of exercise (fig. 20, lower panel). Because about 25% of body heat loss comes from the head, reduced temple skin blood velocity indicates reduced heat transport in this region.

Mean (±SE) thigh skin blood velocity from an active area during exercise was constant at rest and varied from 0.26 ± 0.2 to 0.50 ± 0.11 Hz $\cdot 10^2$ (fig. 21, upper panel). Unlike temple skin response, thigh skin velocity with three treatments increased within 5 min of the start of exercise and all treatment velocities increased to reach, again after about 55 min of exercise, equilibrium levels between 0.61 ± 0.11 and 0.93 ± 0.42 Hz $\cdot 10^2$. Treatment P2GP1 had the lowest (0.22 ± 0.16 Hz $\cdot 10^2$) and 0P1 the highest (0.64 ± 0.39 Hz $\cdot 10^2$) increase in thigh skin velocity at 70 min of exercise (fig. 21, lower panel); in fact, 0P1 blood velocity was elevated appreciably throughout the exercise period.

Salient Responses from Each Treatment

P1P1

1. Significant increase in plasma volume at rest
2. Showed the only positive exercise urinary free water clearance
3. Lowest change in exercise heart rate
4. Lowest change in exercise rectal temperature

P2P1

1. No effect of double strength [P1] on rest or exercise plasma volume
2. Low exercise urinary volume
3. Highest positive water balance at rest
4. Greatest negative exercise water balance

HAP1

1. Significant increase in plasma volume at rest
2. Highest level of exercise plasma volume
3. Highest level of rest and exercise plasma sodium, potassium, and osmotic content
4. Lowest plasma glucose concentration and content at rest
5. High exercise plasma glucose content in spite of no glucose intake
6. High exercise urinary volume
7. Highest rest and exercise urinary sodium excretion
8. Highest exercise urinary potassium and osmotic excretion
9. Lower rest and exercise urinary free water clearance
10. Greatest change (increase) in exercise heart rate
11. Least change in exercise thigh sweat rate
12. Showed the only negative water balance at rest
13. Least change in exercise temple skin blood flow

P2GP1

1. No effect of glycerol on rest or exercise plasma volume
2. Higher urinary volume at rest
3. Greatest change (increase) in exercise rectal temperature
4. Least change in exercise thigh skin blood flow

0P1

1. Compared with no drinking, P1 increased plasma volume
2. Highest exercise plasma glucose content
3. Low rest and exercise urinary volume
4. Lower rest and exercise urinary free water clearance
5. Greatest change (increase) in exercise heart rate
6. Greatest change (increase) in exercise forearm sweat rate
7. Greatest change (increase) in exercise thigh sweat rate
8. Greatest change (increase) in exercise temple skin blood flow
9. Greatest change in exercise thigh skin blood flow

00

1. Low rest and exercise urinary volume
2. Lower rest and exercise urinary free water clearance
3. Least change (increase) in exercise forearm sweat rate

Conclusion

HyperAde (164 mEq/L Na^+), with the lowest osmolality of the four fluid formulations, maintained plasma volume at rest and during exercise, whereas the other formulations with low Na^+ and higher osmolality (365 to 1,382 mOsm/kg) did not. However, Performance 1 increased plasma volume at rest. Thus, drink composition appears to be more important than drink osmolality for increasing plasma volume at rest and for maintaining it during moderately heavy submaximal exercise.

References

Adolph, E. F.; and Associates: Physiology of Man in the Desert. New York: Interscience, 1947.

Bartlett, F.: Psychological Criteria of Fatigue. In Symposium on Fatigue, edited by W.F. Floyd and A.T. Welford. London: H. K. Lewis, 1953, pp. 1–5.

Booth, D. A.: Influences on Human Fluid Consumption. In Thirst : Physiological and Psychological Aspects, edited by D. J. Ramsay and D. Booth. Chap. 4. New York: Springer-Verlag, 1991, pp. 53–73.

Campbell, T. J.; Frohman, B.; and Reeve, E. B.: A Simple, Rapid, and Accurate Method of Extracting T-1824 from Plasma Adapted to the Routine Measurement of Blood Volume. J. Lab. Clin. Med. 52(5): 768–777, 1958.

Castle, B. L.; and Greenleaf, J. E.: Exercise Temperature Regulation in Man During Hypohydration and Hyperhydration. J. Appl. Physiol. 30: 847–853, 1971.

Fitts, R. H.: Cellular Mechanisms of Muscle Fatigue. Physiol. Rev. 74(1): 49–94, 1994.

Gisolfi, C. V.: Use of Electrolytes in Fluid Replacement Solutions: What Have We Learned from Intestinal Absorption Studies? In Fluid Replacement and Heat Stress, edited by B.M. Marriott. Washington, D.C.: National Academy Press, 1991, pp. 11–21.

Greenleaf, J. E.: Effects of Dehydration on Performance in Man : Annotated Bibliography. NASA TM X-62,308, 1973.

Greenleaf, J. E.: Hyperthermia and Exercise. In Int. Rev. Physiol., vol. 20, Environmental Physiology III, edited by D. Robertshaw. Baltimore: University Park Press, 1979, pp. 157–208.

Greenleaf, J. E.: The Consequences of Exercise on Thirst and Fluid Intake. In Thirst: Physiological and Psychological Aspects, edited by D. J. Ramsay and D. Booth, Chap. 27. New York: Springer-Verlag, 1991, pp. 412–421.

Greenleaf, J. E.: Problem: Thirst, Drinking Behavior, and Involuntary Dehydration. Med. Sci. Sports Exerc. 24(6): 645–656, 1992.

Greenleaf, J. E.; and Castle, B. L.: External Auditory Canal Temperature as an Estimate of Core Temperature. J. Appl. Physiol. 32: 194–198, 1972.

Greenleaf, J. E.; Convertino, V. A.; and Mangseth, G. R.: Plasma Volume During Stress in Man: Osmolality and Red Cell Volume. J. Appl. Physiol. 47: 1031–1038, 1979.

Greenleaf, J. E.; Geelen, G.; Jackson, C. G. R.; Saumet, J.-L.; Juhos, L. T.; Keil, L. C.; Fegan-Mayer, D.; Dearborn, A.; Hinghofer-Szalkay, H.; and Whittam, J. H.: Vascular Uptake of Rehydration Fluids in Hypohydrated Men at Rest and Exercise. NASA TM 103942, 1992.

Greenleaf, J. E.; and Williams, B. A.: Thermistor Holder for Skin Temperature Measurements. U.S. Patent 3,983,753; NASA-CASE-ARC-10855-1, 1976.

Hardy, J. D.; and Dubois, E. F.: Technic of Measuring Radiation and Convection. J. Nutr. 15: 461–475, 1938.

Kozlowski, S.; Greenleaf, J. E.; Turlejaska, E.; and Nazar, K.: Extracellular Hyperosmolality and Body Temperature During Physical Exercise in Dogs. Am. J. Physiol. 239: R180–R183, 1980.

Luetkemeier, M. J.; and Thomas, E. L.: Hypervolemia and Cycling Time Trial Performance. Med. Sci. Sports Exerc. 26(4): 503–509, 1994.

Marriott, B. M. (editor): Fluid Replacement and Heat Stress. Washington D.C.: National Academy Press, 1994.

Maughan, R. J.; and Noakes, T. D.: Fluid Replacement and Exercise Stress. A Brief Review of Studies on Fluid Replacement and Some Guidelines for the Athlete. Sports Med. 12(1): 16–31, 1991.

Murray, R.: The Effects of Consuming Carbohydrate-Electrolyte Beverages on Gastric Emptying and Fluid Absorption During and Following Exercise. Sports Med. 4(5): 322–351, 1987.

Pitts, G. C.; Johnson, R. E.; and Consolazio, F. C.: Work in the Heat as Affected by Intake of Water, Salt and Glucose. Am J. Physiol. 142: 253–259, 1944.

Rothstein, A.; Adolph, E. F.; and Wills, J. H.: Voluntary Dehydration. In Physiology of Man in the Desert, edited by E. F. Adolph and Associates. New York : Interscience, 1947, pp. 254–270.

Spaul, W. A.: Physiological Effects of Simultaneous Exposures to Heat and Vibration. Ph.D. Thesis, Univ. of Calif., Berkeley; NASA TM 84400, 1983.

Table 1. Anthropometric and peak exercise data for the six subjects

	Anthropometric data							Peak exercise data						
									Ventilation					
Subject	Age, yr	Height, cm	Weight, kg	Surface area, m2	Plasma volume, mL	Blood volume, mL	Blood volume, mL/kg	Load, kg-m/min	STPD, L/min	BTPS, L/min	Heart rate, b/min	Oxygen, L/min	Uptake, mL/min/kg	Respiratory exchange ratio
CAL	24	186	67.20	1.90	3240	5598	83	1400	104.24	125.50	193	2.64	39	1.34
DUW	39	192	97.74	2.28	4112	7373	75	1700	109.85	132.04	162	2.92	30	1.11
GUF	36	170	57.42	1.66	2551	4620	80	1500	98.60	118.71	170	2.61	45	1.33
PAU	23	182	89.20	2.11	2899	5338	60	1700	123.85	148.62	199	3.55	40	1.24
PED	22	181	63.72	1.82	3215	5591	88	1800	85.91	103.09	210	3.56	56	1.19
REA	34	183	85.75	2.08	2729	4615	54	1200	106.50	128.01	187	2.64	31	1.27
$\overline{X}$	30	182	76.84	1.98	3124	5522	73	1550	104.83	126.00	187	2.99	40	1.25
±SD	8	7	16.19	0.22	505	923	12	226	12.54	15.04	17	0.45	10	0.09
±SE	3	3	6.61	0.09	206	377	5	92	5.12	6.14	7	0.19	4	0.04

STPD = standard temperature, pressure, dry.
BTPS = body temperature, pressure, saturated.

Table 2. Drink composition per 2000 mL (package label data)

	P1[b]	P2[c]	P2G[d]	HA[e]
Sodium chloride (gm)	–	–	–	9.00
Sodium Citrate (gm)	3.87	7.74	7.74	15.44
Dextrose (gm)	41.12	82.24	82.24	–
Aspartame (gm)	–	–	–	0.72
Glycerol (gm)	–	–	100.87	–
Shaklee Performance[a] (gm)	222.28	444.56	444.56	–
Total	222.28	444.56	444.56	25.16
Total volume (mL)	2,000	2,000	2,000	2,000
Ionic concentration: (mEq/L, % weight/volume)				
Na^+	19.61/0.04	39.22/0.09	39.22/0.09	157/0.36
K+	5.01/0.02	10.02/0.04	10.02/0.04	–
Cl^-	4.98/0.02	9.96/0.04	9.96/0.04	76/0.27
Mg^{++}	0.40/0.01	0.80/0.01	0.80/0.01	–
Ca^{++}	1.96/0.02	3.92/0.03	3.92/0.03	–
P^{++++}	0.51/0.01	1.02/0.02	1.02/0.02	–
Total	32.47/0.11	69.94/0.22	69.94/0.22	233/0.63
Carbohydrate (% weight/volume)				
Glucose	1.85	3.70	3.70	–
Fructose	2.43	4.85	4.85	–
Maltodextrin	5.44	10.88	10.88	–
Total	9.72	19.43	19.43	–
Measured drink solute concentrations				
Na^+ (mEq/L)	55.2	97.1	112.7	163.7
K^+ (mEq/L)	5.3	10.3	10.7	<0.1
Osmolality (mOsm/kgH_2O)	365	791	1382	253
Glycerol (mg/dL)	2.0	4.0	2916	1.0
Glucose (mg/dL)	2049	3579	3543	<0.5
Citrate (mg/dL)	416	753	731	854

[a]Shaklee U.S., Inc., San Francisco, CA 94111.
[b]Shaklee Performance.
[c]Double-strength Shaklee Performance.
[d]Double-strength Shaklee Performance plus 80 mL glycerol.
[e]HyperAde –NaCl/Na citrate (0.036% Na^+)

Table 3. Individual drink volume (10 mL/kg body weight) for the rest and exercise phases

Drink / Subject	P1P1	P2P1	P2GP1	HAP1	0P1	0
CAL	1,342	1,342	1,318	1,346	656	0
DUW	1,978	2,018	2,000	1,984	977	0
GUF	1,112	1,092	1,106	1,102	561	0
PAU	1,800	1,785	1,794	1,812	892	0
PED	1,264	1,274	1,266	1,268	627	0
REA	1,708	1,696	1,722	1,720	866	0
$\overline{X}$	1,534	1,535	1,534	1,539	796	0
±SD	342	353	353	348	164	0
±SE	140	144	144	142	67	0

Table 4. Individual subject rest and submaximal exercise data[a]

Subject	$\dot{V}O_2$ rest, L/min	Load,[a] kg-m/min	$\dot{V}O_2$ exercise,[a] L/min	$\dot{V}O_2$ exercise,[a] % peak	$\dot{V}O_2$ exercise,[a] mL/min · kg
CAL	0.34	700	1.80	68	27
DUW	0.44	900	2.43	83	25
GUF	0.31	700	1.70	65	30
PAU	0.44	900	2.46	69	28
PED	0.38	1000	2.19	62	34
REA	0.40	700	1.90	72	22
$\overline{X}$	0.39	817	2.08	70	28
±SD	0.05	133	0.33	7	4
±SE	0.02	54	0.13	3	2

[a] $\overline{X}$ of six treatments.

$\dot{V}O_2$ = oxygen uptake.

Table 5. Mean environmental parameters for the six treatments

Variable		P1P1	P2P1	P2GP1	HAP1	0P1	00	Mean
				Rest phase				
Dry bulb temperature (°C)	$\overline{X}$	22.2	21.9	21.4	21.8	22.1	21.4	21.8
	±SD	0.9	1.1	0.5	0.8	0.6	0.2	0.3
	±SE	0.4	0.4	0.2	0.3	0.3	0.1	0.1
Relative humidity (%)	$\overline{X}$	50	48	45	48	52	51	49
	±SD	5	5	1	4	6	2	3
	±SE	2	2	1	2	3	1	1
Wind speed (ft/min)	$\overline{X}$	23	16	25	25	29	22	23
	±SD	8	8	8	7	12	5	4
	±SE	3	3	3	3	5	2	2
Barometric pressure (mmHg)	$\overline{X}$	764.3	764.3	763.1	764.1	762.5	763.5	763.6
	±SD	1.0	2.0	1.8	0.7	0.9	2.2	0.7
	±SE	0.4	0.8	0.8	0.3	0.4	0.9	0.3
				Exercise phase				
Dry bulb temperature (°C)	$\overline{X}$	22.2	21.8	21.0	21.6	22.2	21.9	21.8
	±SD	0.8	1.2	0.3	0.5	0.6	0.3	0.4
	±SE	0.3	0.5	0.1	0.2	0.2	0.1	0.2
Relative humidity (%)	$\overline{X}$	49	49	46	51	52	51	50
	±SD	5	2	3	1	5	3	2
	±SE	2	1	1	1	2	1	1
Wind speed (ft/min)	$\overline{X}$	53	60	49	54	52	52	53
	±SD	6	15	5	1	6	6	4
	±SE	3	6	2	1	3	3	2
Barometric pressure (mmHg)	$\overline{X}$	764.0	764.1	763.2	764.0	762.2	763.7	763.5
	±SD	1.2	1.7	1.9	0.8	1.0	2.4	0.7
	±SE	0.5	0.7	0.8	0.3	0.4	1.0	0.3

Rest phase data are averages of –65- and –35-min values; exercise phase data are averages of 30- and 60-min values.

Table 6. Mean (±SE) urine electrolyte concentrations for the rest (–105 to –15 min) and exercise (–15 to +10 min) phases for the six treatments

Variable	P1P1	P2P1	P2GP1	HAP1	0P1	00
		Rest phase				
Urine Na^+ (μEq/L)	63.3	65.8	81.1	100.4	113.1	111.2
	(17.4)	(17.5)	(22.8)	(18.6)	(17.7)	(21.7)
Urine K^+ (μEq/L)	18.2	17.8	29.0	39.8	51.7	66.8
	(4.1)	(4.5)	(8.0)	(8.2)	(14.1)	(22.1)
Osmolality (mOsm/kgH_2O)	328	368	443	498	752	712
	(56)	(62)	(121)	(79)	(146)	(135)
		Exercise phase				
Urine Na^+ (μEq/L)	47.6	72.9	55.1	80.6	102.3	126.5
	(8.6)	(22.4)	(7.8)	(19.9)	(12.2)	(18.9)
Urine K^+ (μEq/L)	27.4	53.6	27.1	58.5	85.9	90.2
	(3.8)	(24.5)	(2.1)	(6.2)	(13.4)	(19.2)
Osmolality (mOsm/kgH_2O)	280	451	397	442	781	843
	(33)	(124)	(87)	(89)	(116)	(105)

Table 7. Mean (±SE) water balance, respiratory water loss, insensible water loss, and sweat rate for the rest (–105 to –15 min) and exercise (–15 to 70 min) phases for the six treatments

Variable	P1P1	P2P1	P2GP1	HAP1	0P1	00
		Rest phase				
Water balance, g/m^2 · hr	47	93	1	–78	6	–23
	(19)	(52)	(23)	(47)	(39)	(65)
Respiratory water loss, g/m^2 · hr	12	12	12	12	10	10
	(1)	(1)	(1)	(1)	(1)	(1)
Insensible water loss, g/m^2 · hr	18	18	18	18	18	18
Sweat rate, g/m^2 · hr	77	123	31	–48	34	–5
	(19)	(52)	(22)	(47)	(39)	(65)
		Exercise phase				
Water balance, g/m^2 · hr	–197	–315	–125	–151	–237	–221
	(42)	(73)	(34)	(31)	(73)	(62)
Respiratory water loss, g/m^2 · hr	49	50	52	49	50	49
	(2)	(3)	(2)	(1)	(4)	(3)
Insensible water loss, g/m^2 · hr	18	18	18	18	18	18
Sweat rate, g/m^2 · hr	130	247	55	84	169	154
	(40)	(72)	(34)	(30)	(71)	(59)

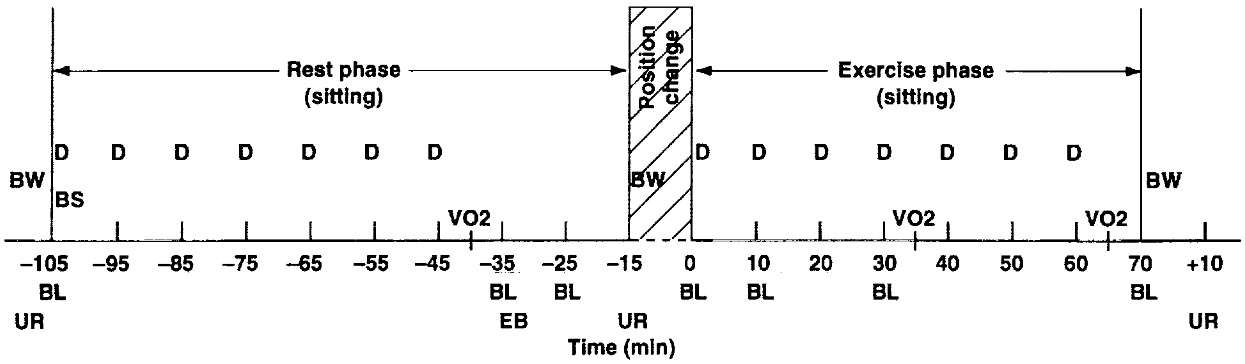

Figure 1. Experimental protocol. UR = urine, BL = blood sample, EB = Evans blue injection, BW = body weight, $\dot{V}O_2$ = oxygen uptake, and D = drinking (1/14 of total volume).

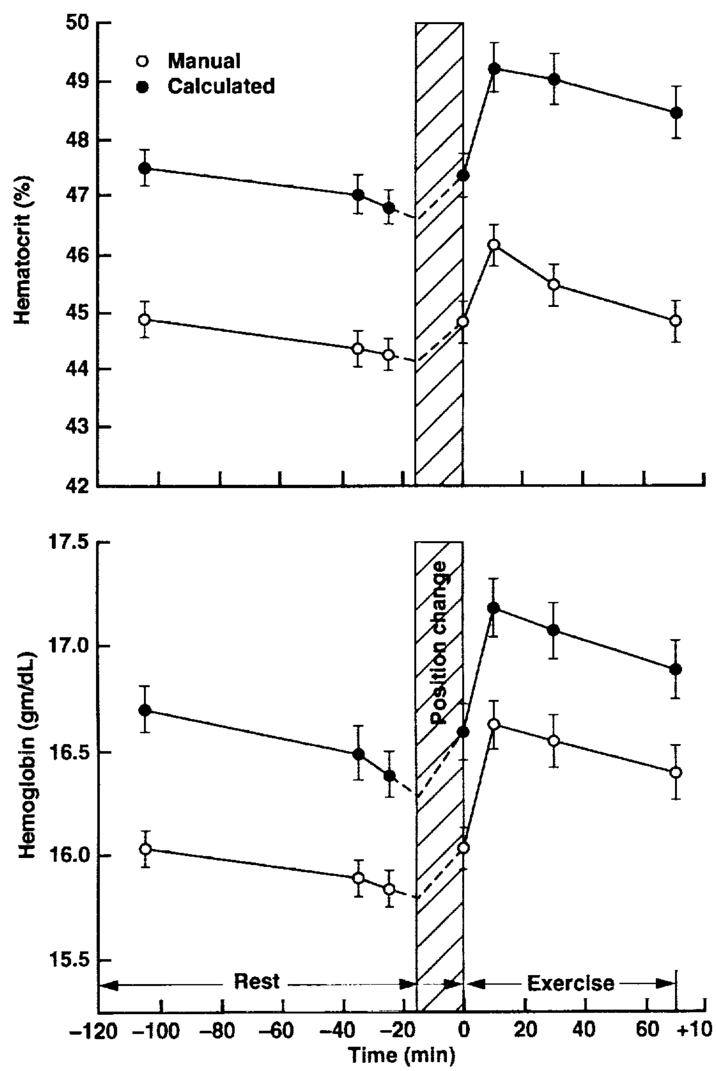

Figure 2. Comparison of manual and automatic (Coulter counter) measurement for hemoglobin and hematocrit at rest and during exercise ($\overline{X}$ ± SE).

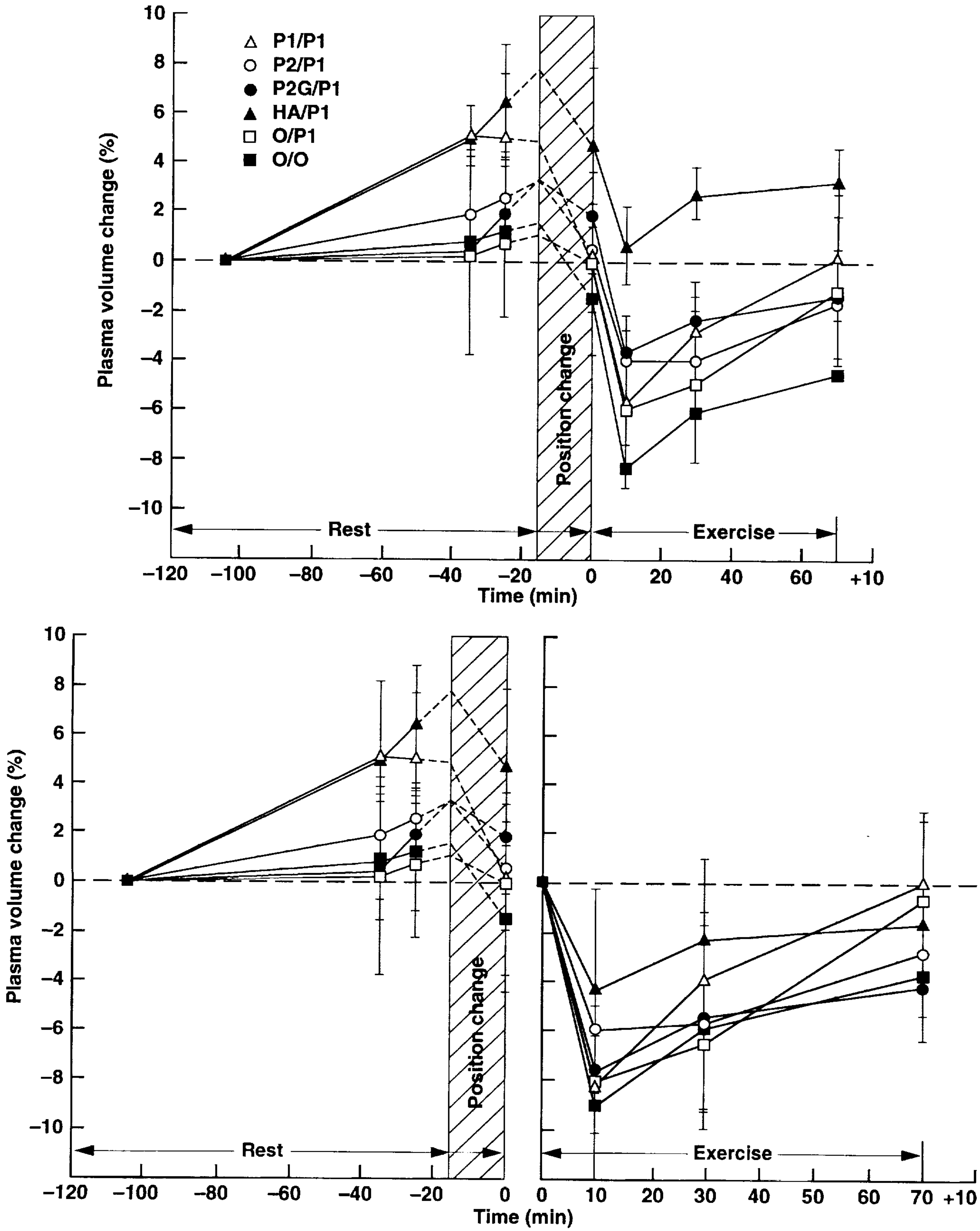

Figure 3. Mean (±SE) change in plasma volume at rest and during exercise for the six treatments.

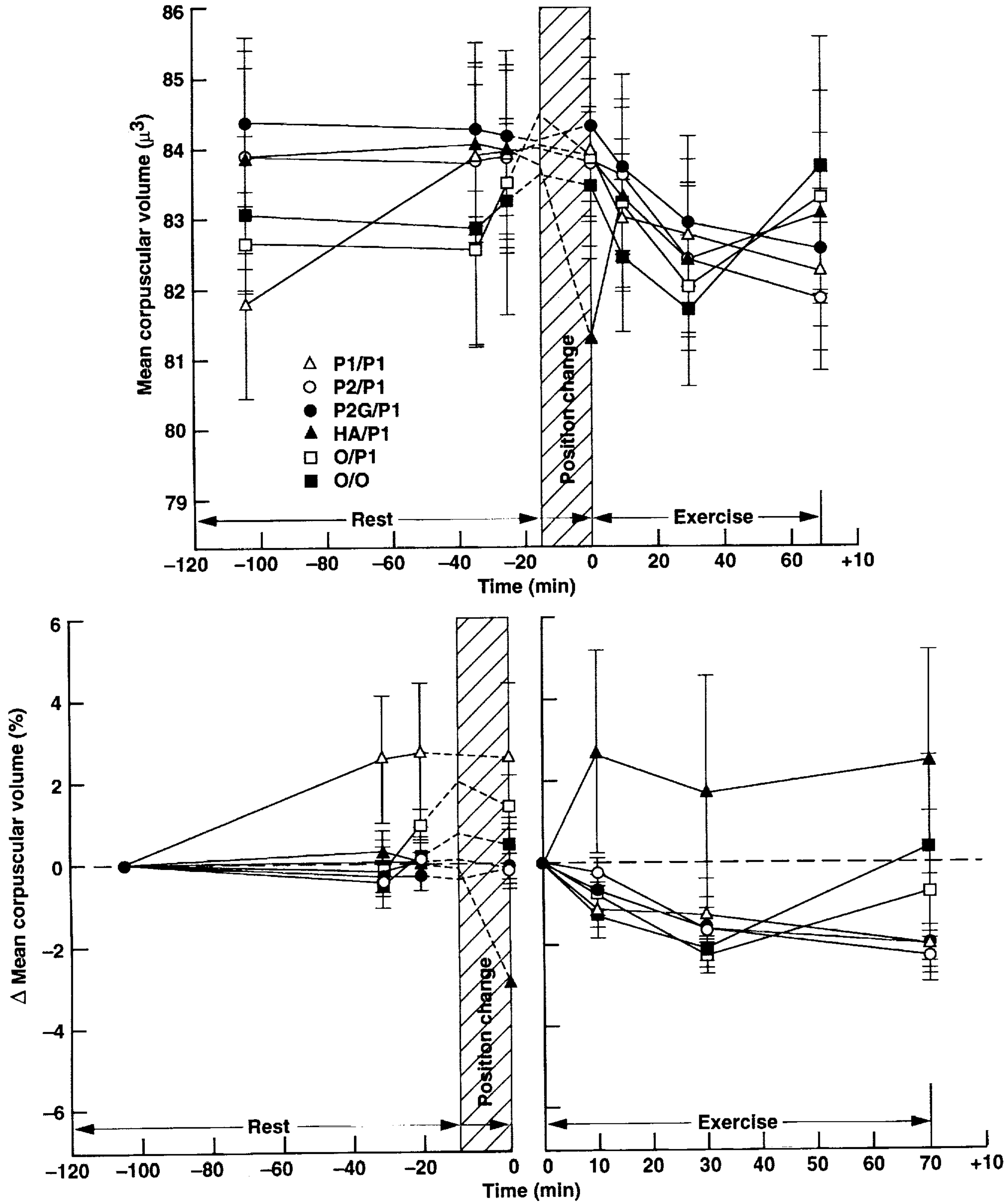

Figure 4. Mean (±SE) mean corpuscular volume at rest and during exercise for the six treatments.

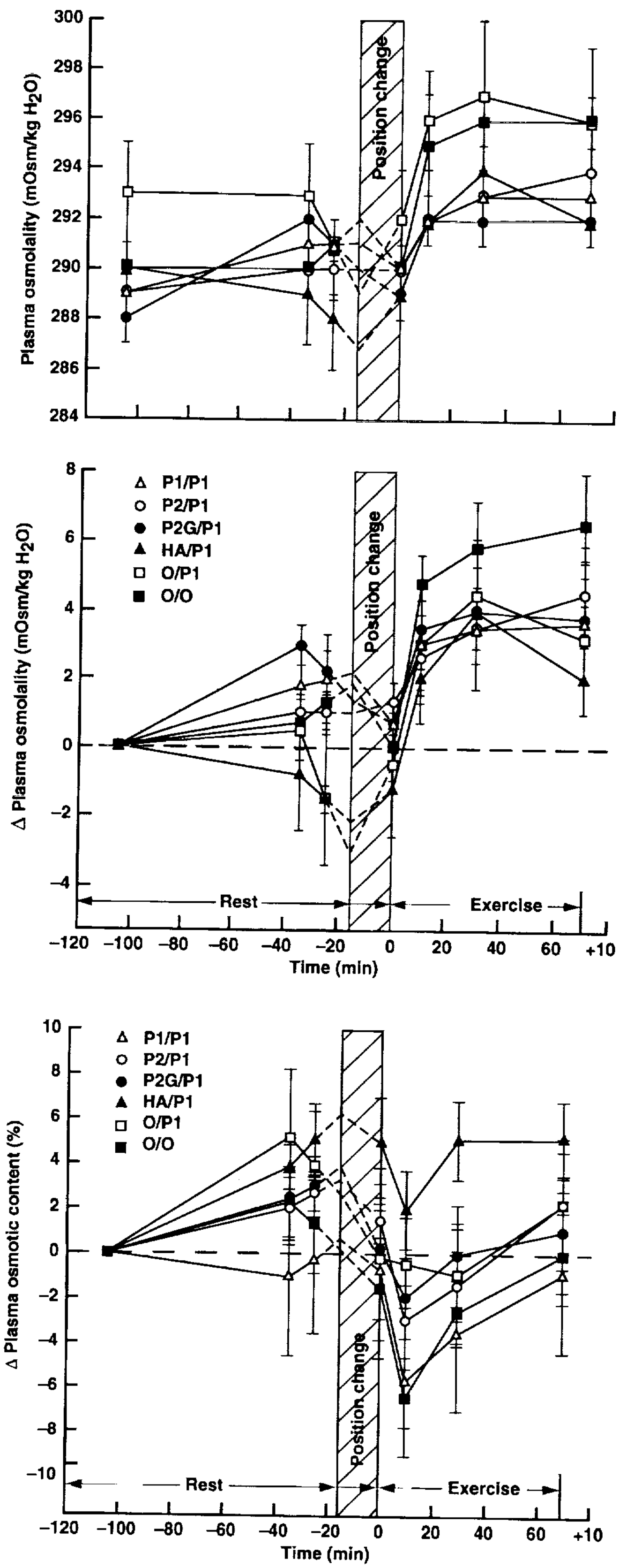

Figure 5. Mean (±SE) plasma osmotic concentration at rest and during exercise for the six treatments.

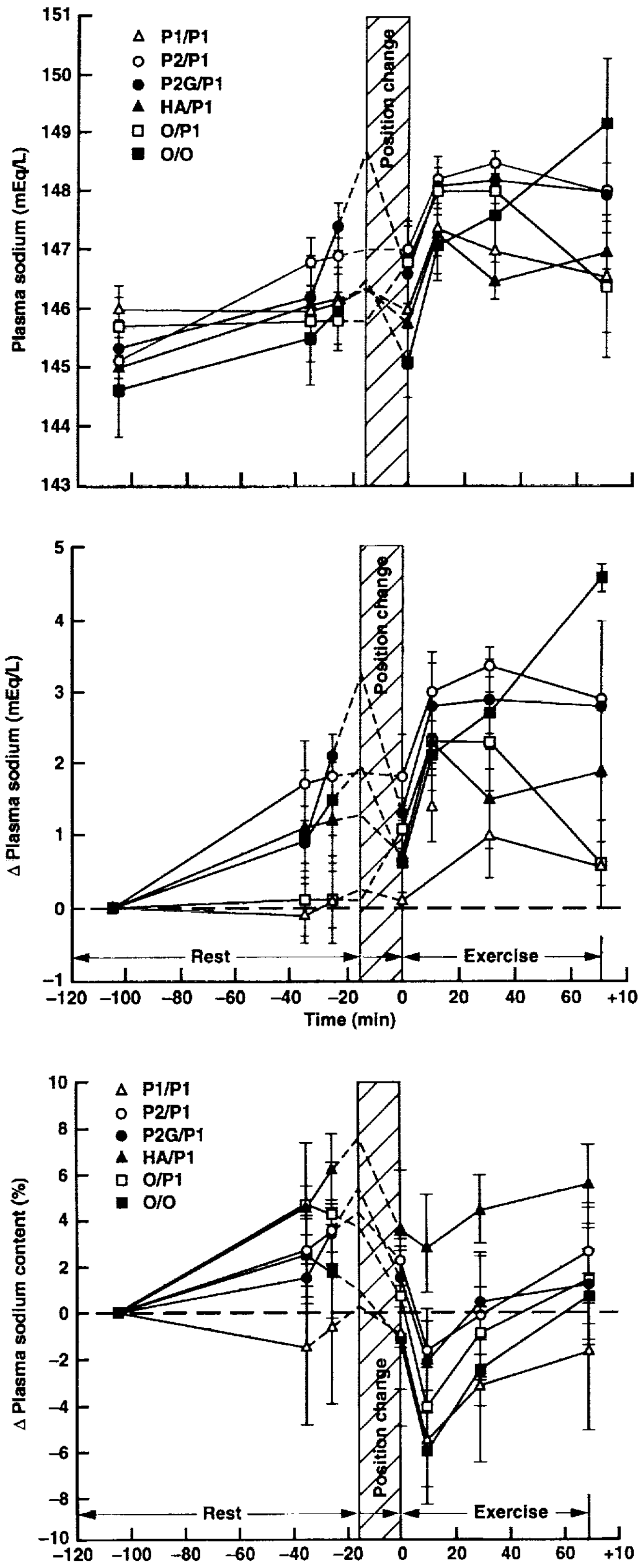

Figure 6. Mean (±SE) plasma sodium concentration at rest and during exercise for the six treatments.

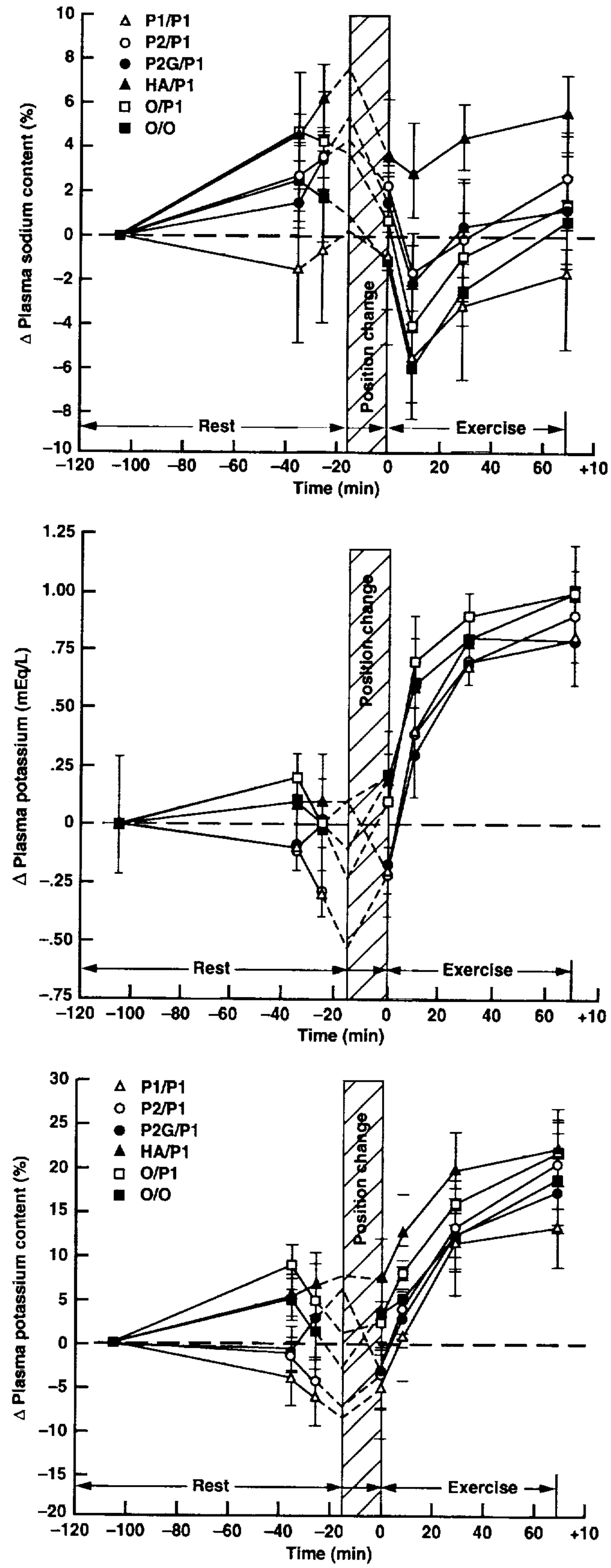

Figure 7. Mean (±SE) plasma potassium concentration at rest and during exercise for the six treatments.

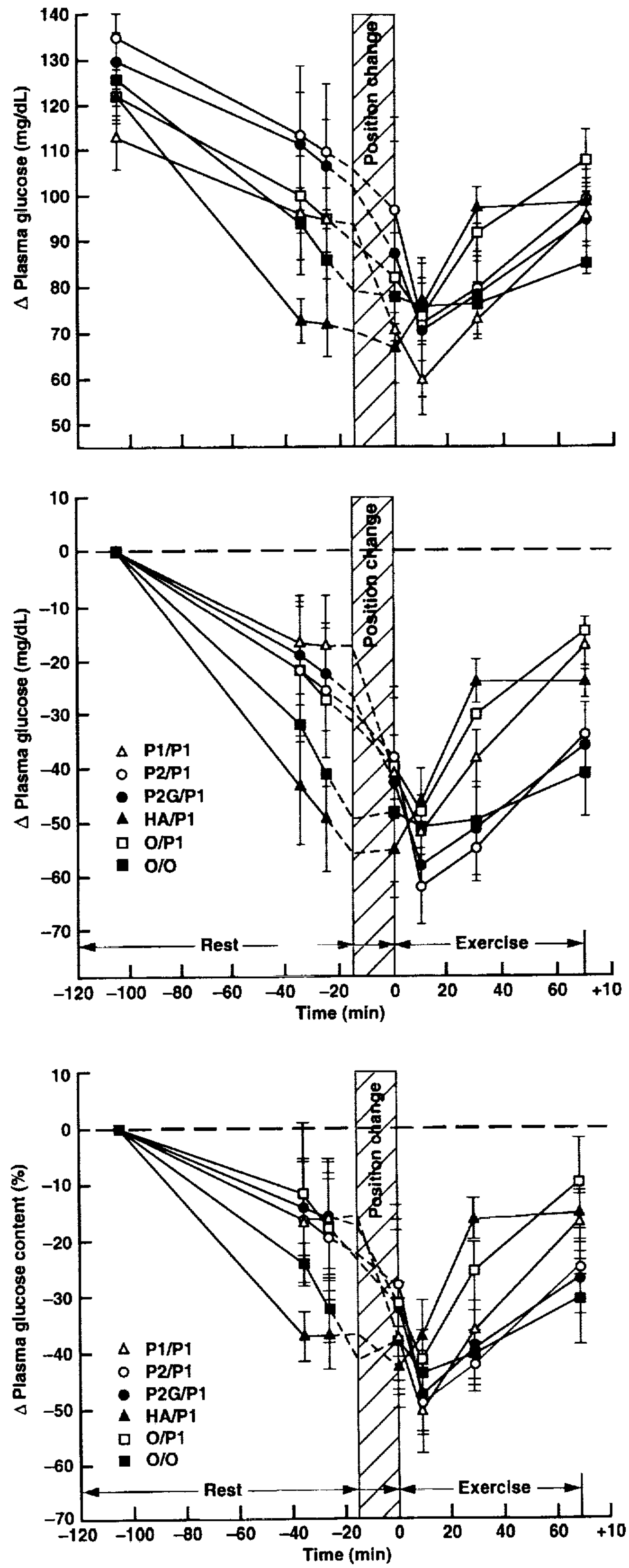

Figure 8. Mean (±SE) plasma glucose concentration at rest and during exercise for the six treatments.

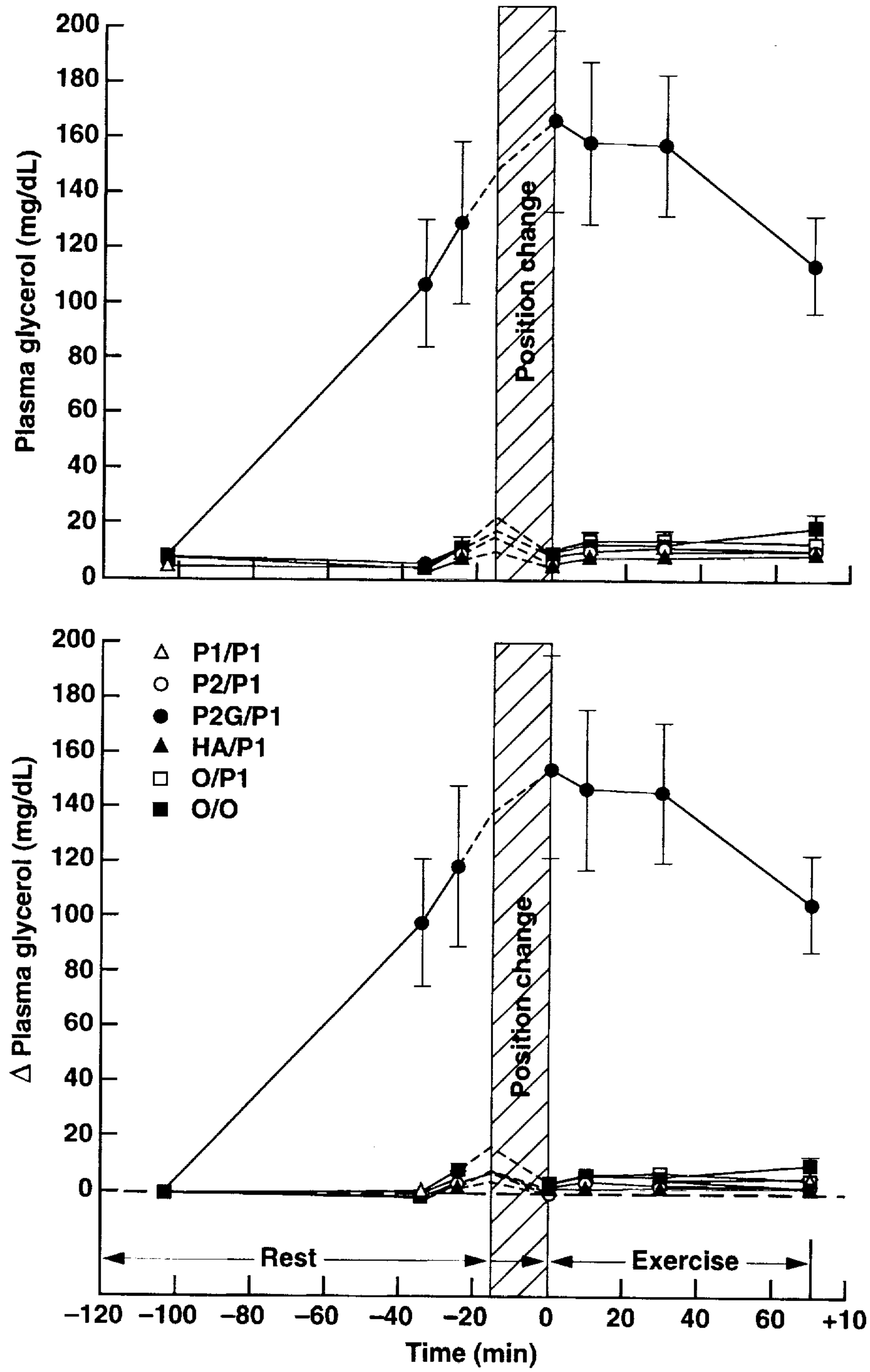

Figure 9. Mean (±SE) plasma glycerol concentration at rest and during exercise for the six treatments.

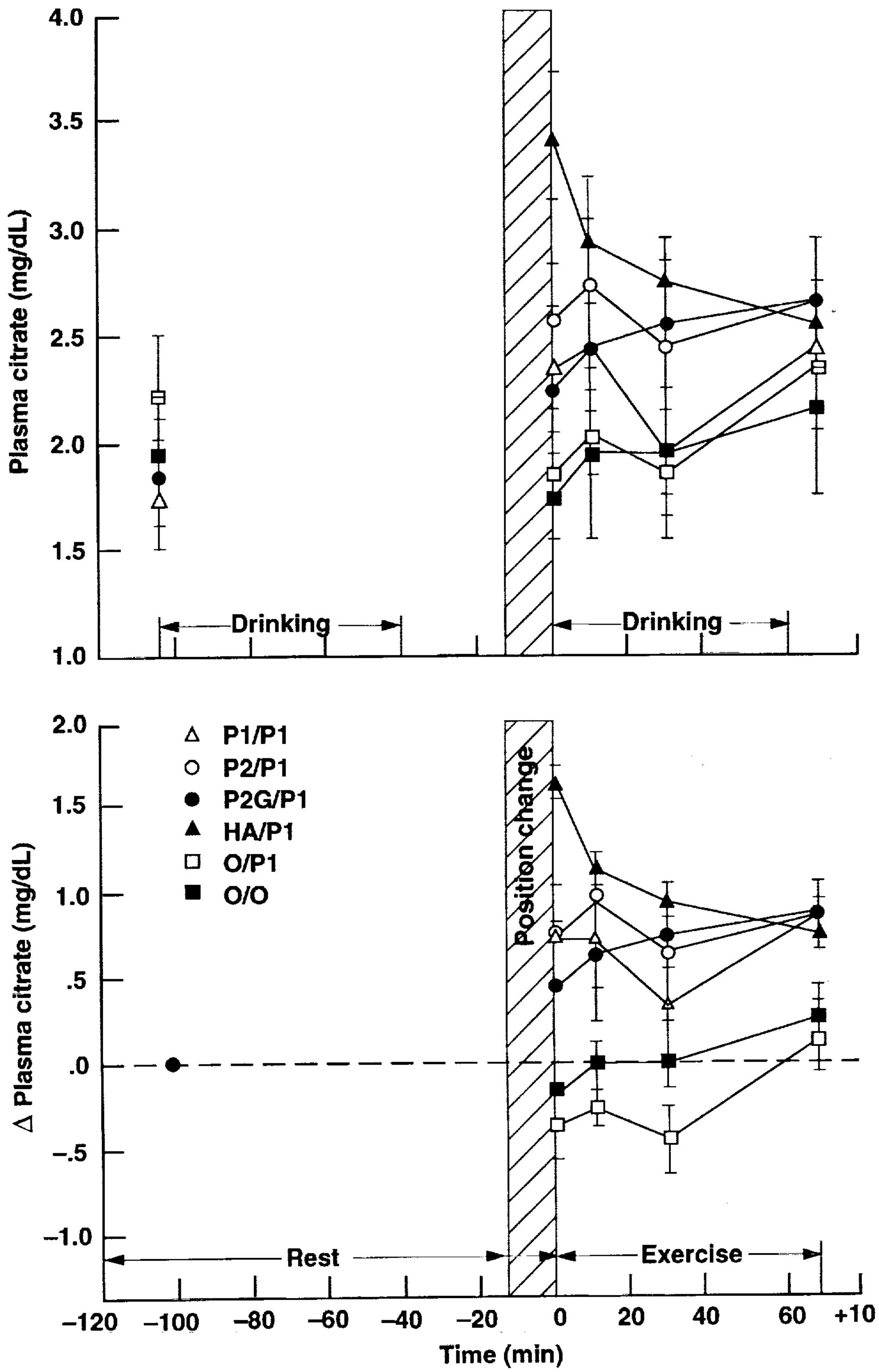

Figure 10. Mean (±SE) plasma citrate concentration at rest and during exercise for the six treatments.

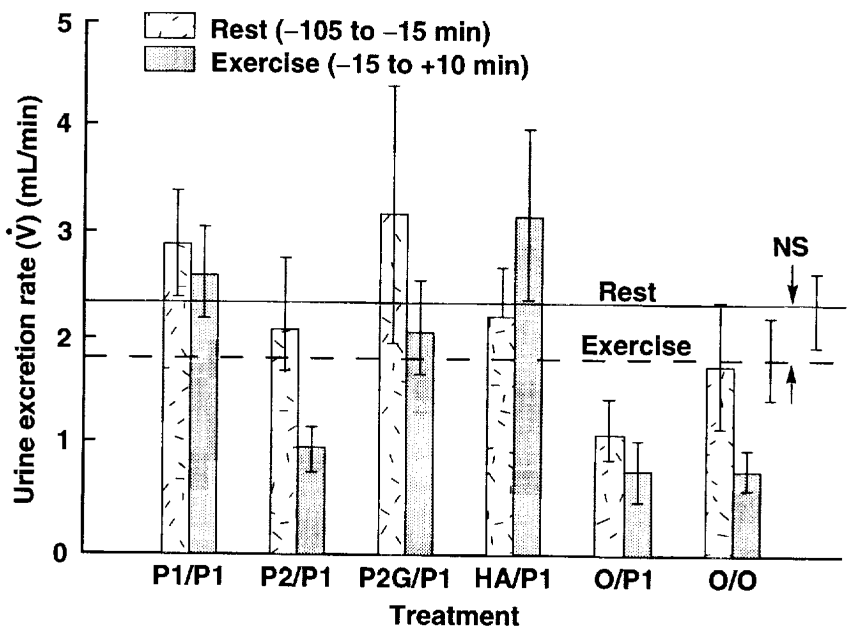

Figure 11. Mean (±SE) urinary excretion rate at rest and during exercise for the six treatments. Solid line is mean (±SE) for rest treatments; dash line is mean (±SE) for exercise treatments.

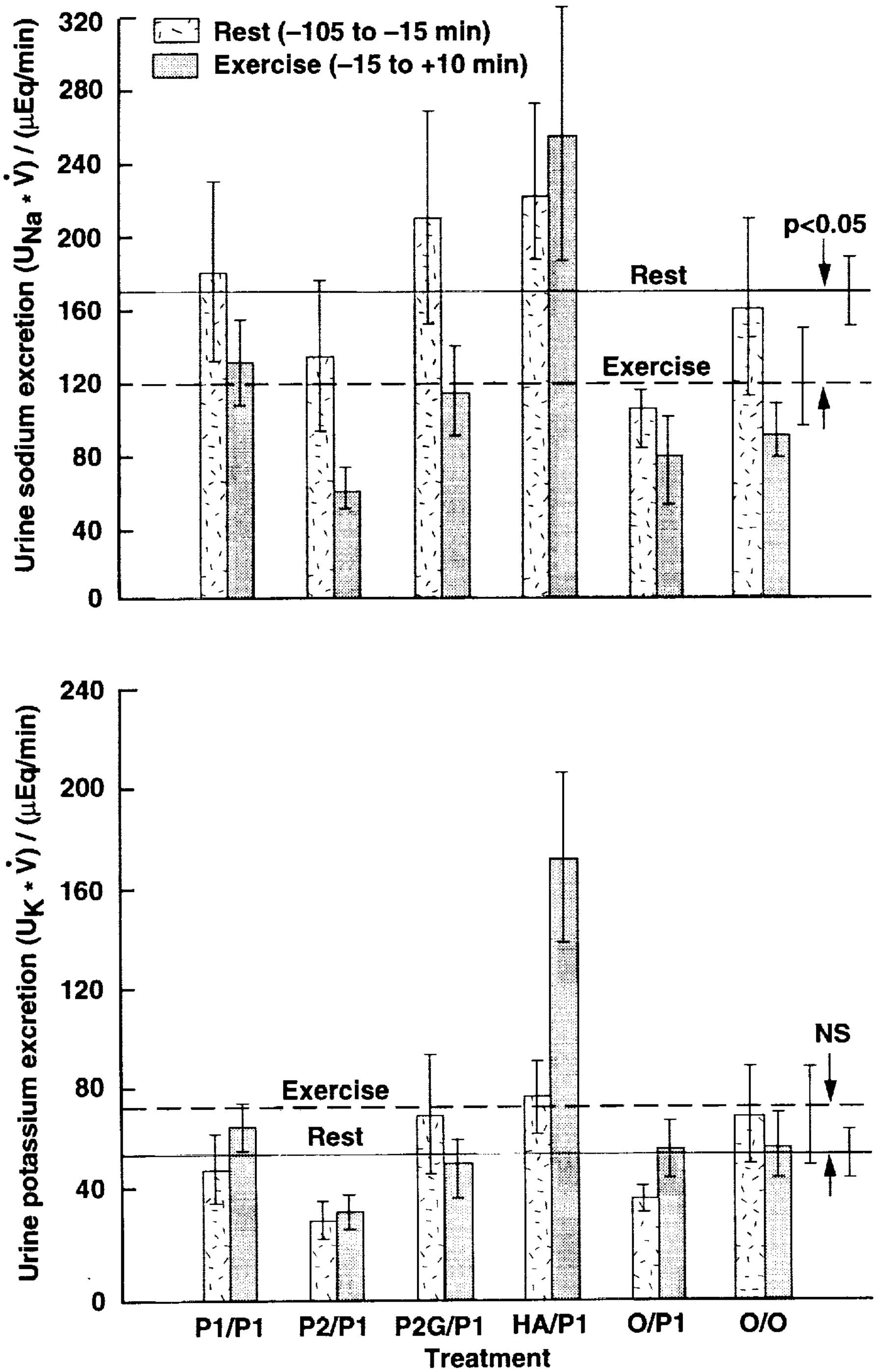

Figure 12. Mean (±SE) urine sodium excretion (upper panel) and potassium excretion (lower panel) at rest and during exercise for the six treatments.

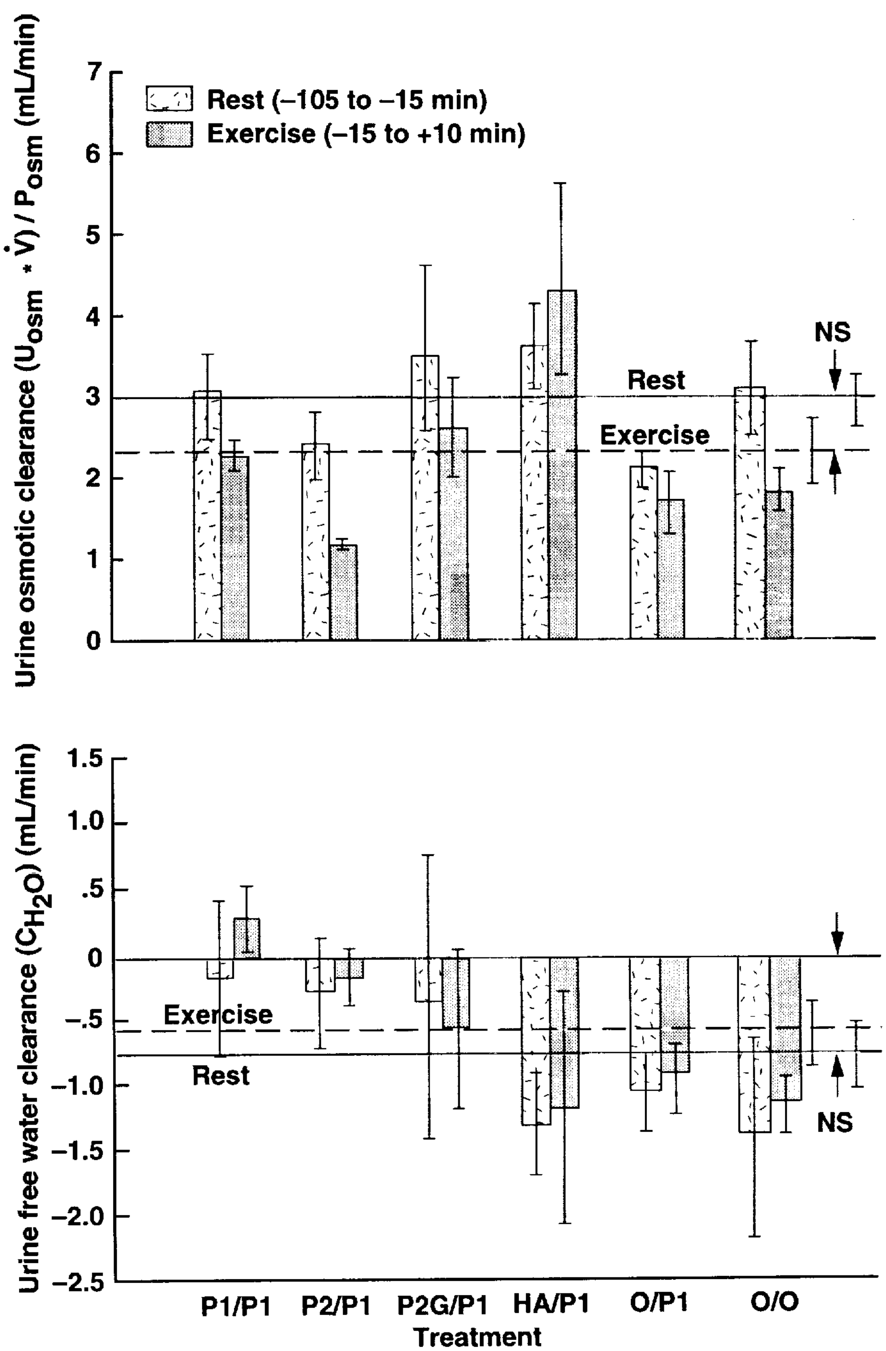

Figure 13. Mean (±SE) urine osmotic clearance (upper panel) and free water clearance (lower panel) at rest and during exercise for the six treatments.

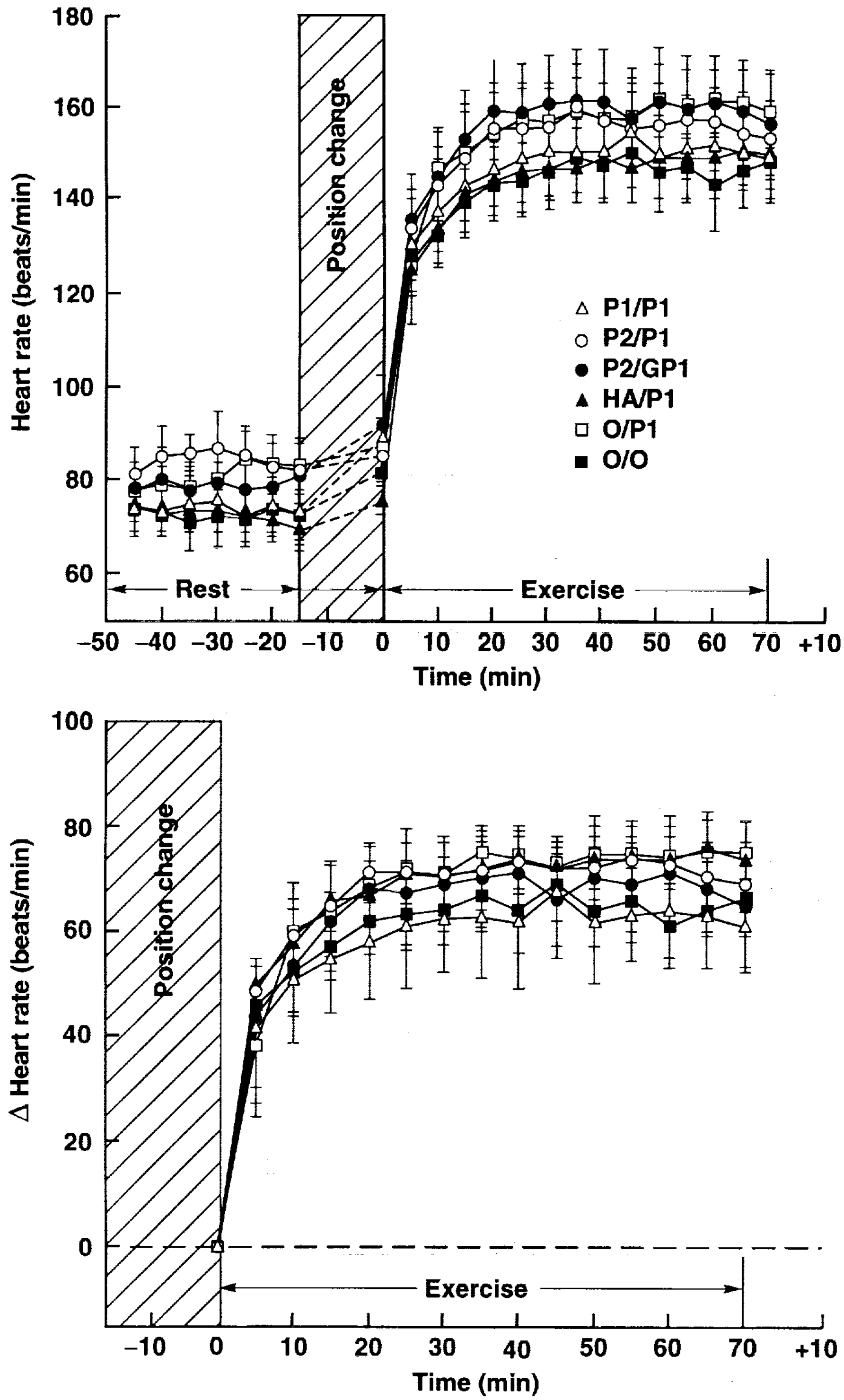

Figure 14. Mean (±SE) heart rate at rest and during exercise for the six treatments.

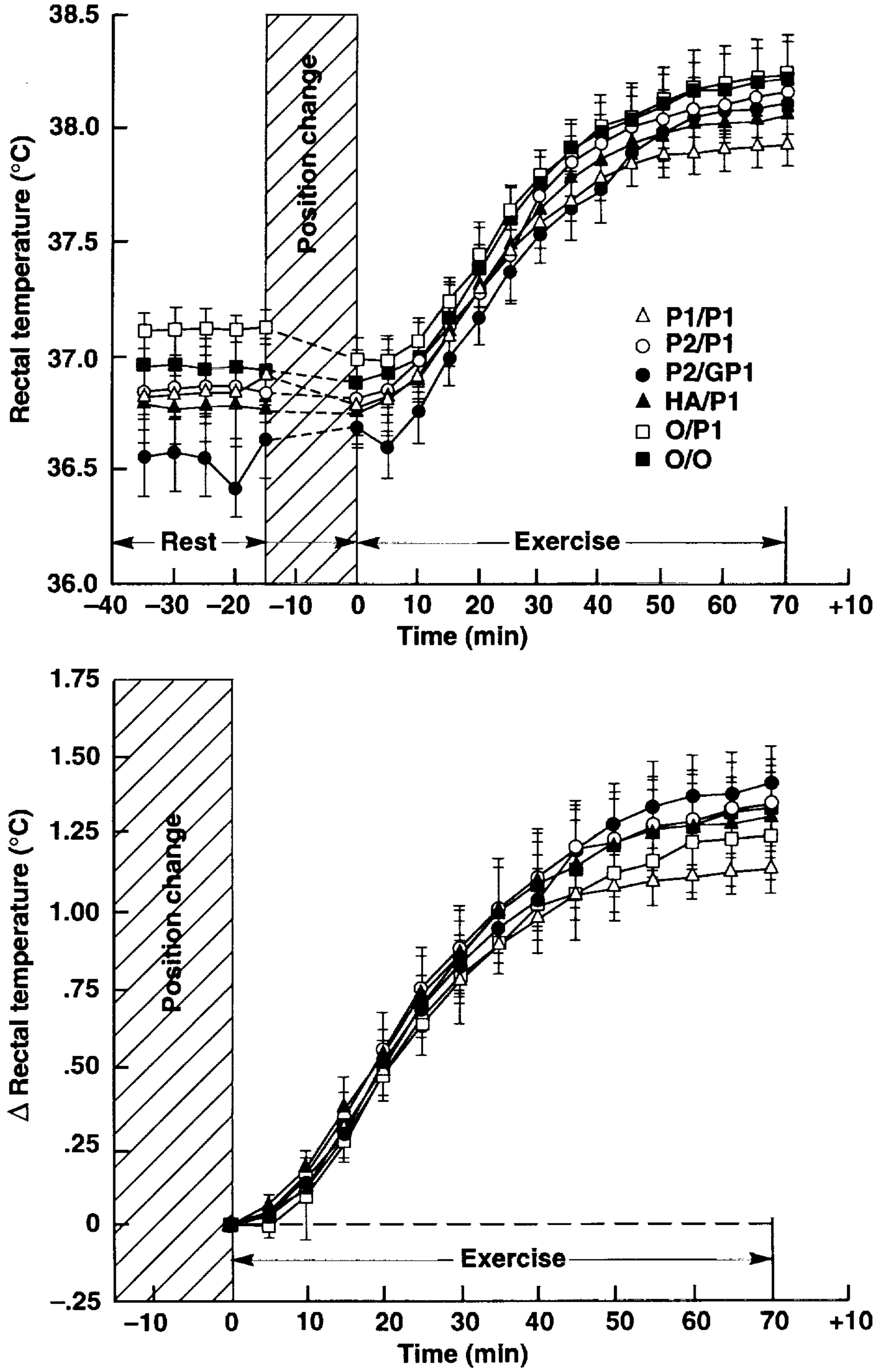

Figure 15. Mean (±SE) rectal temperature at rest and during exercise for the six treatments.

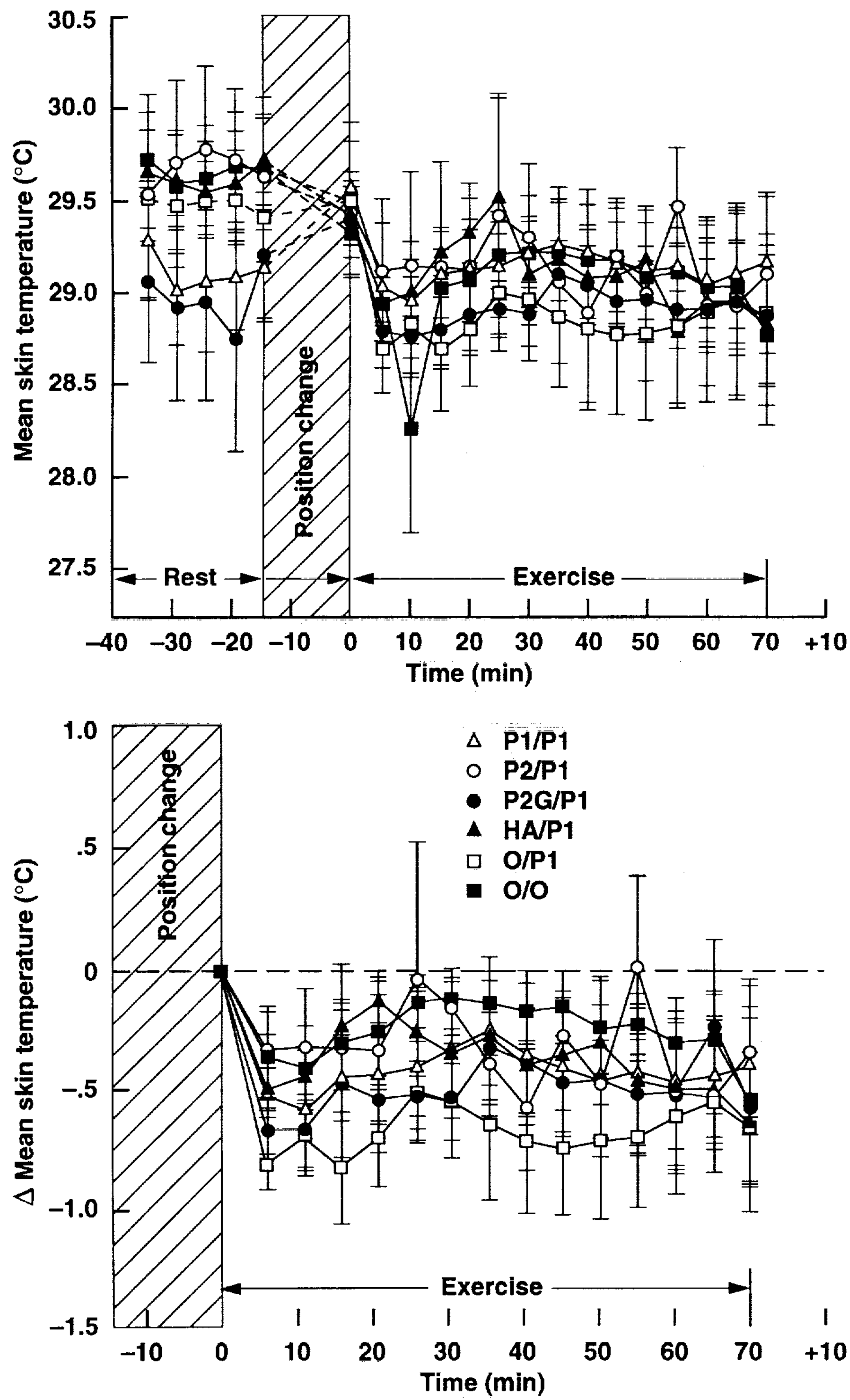

Figure 16. Mean (±SE) mean skin temperature at rest and during exercise for the six treatments.

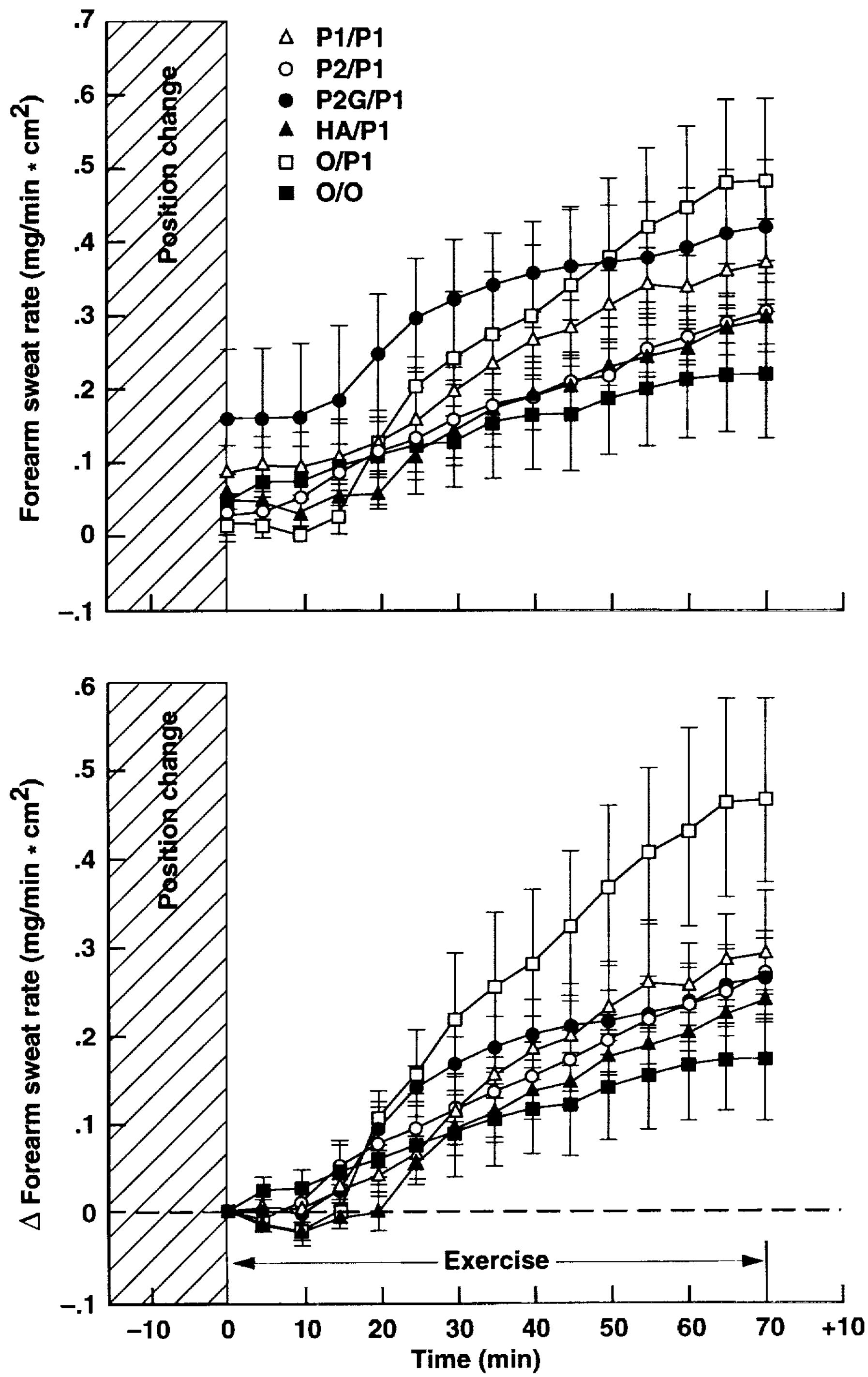

Figure 17. Mean (±SE) forearm sweat rate at rest and during exercise for the six treatments.

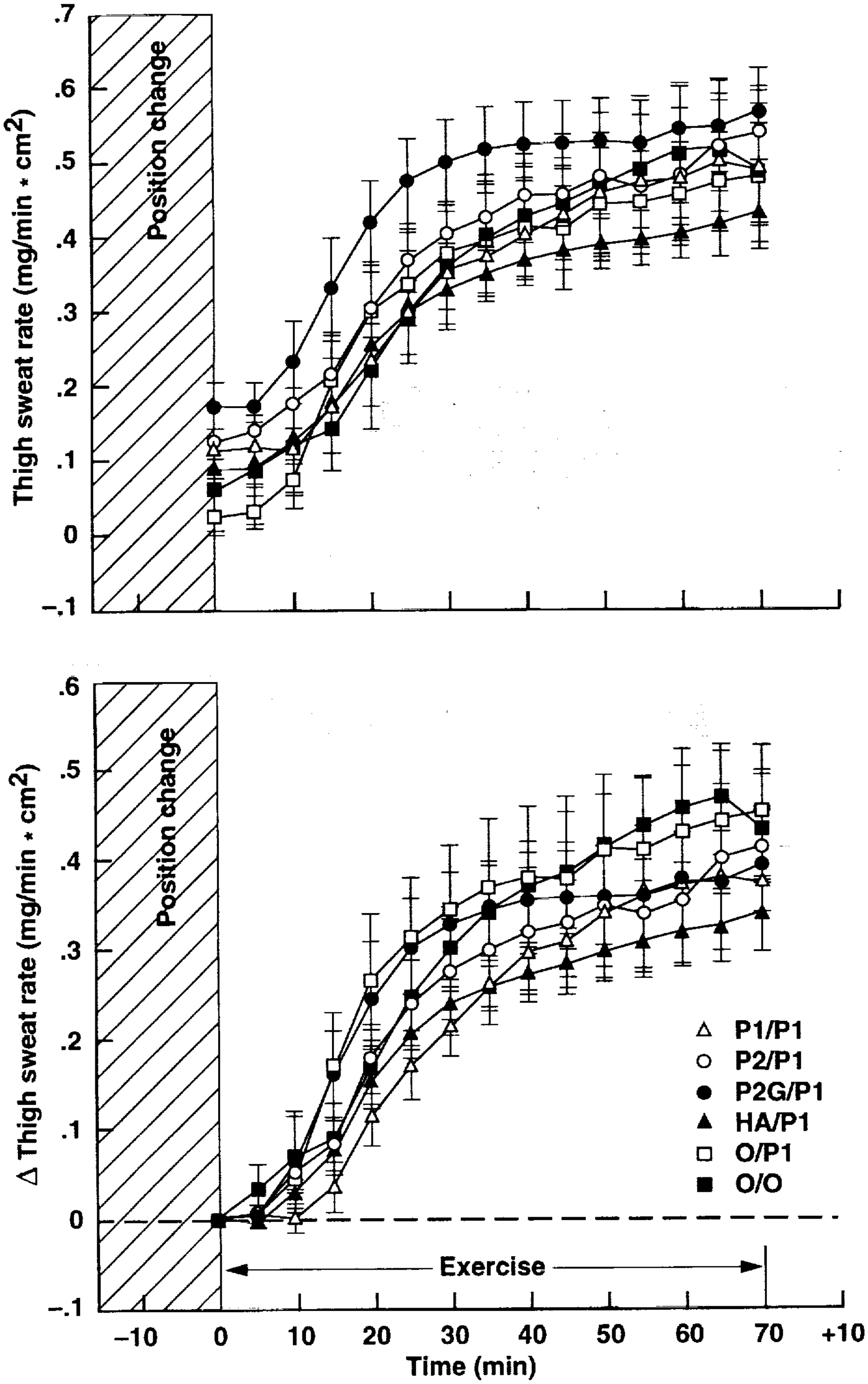

Figure 18. Mean (±SE) thigh sweat rate at rest and during exercise for the six treatments.

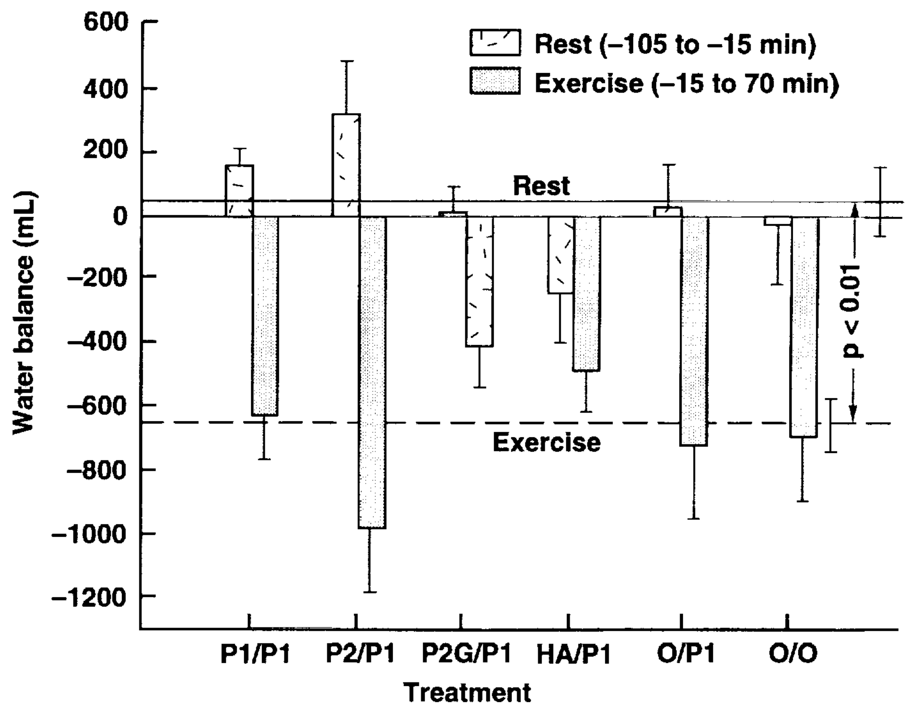

Figure 19. Mean (±SE) water balance at rest and during exercise for the six treatments.

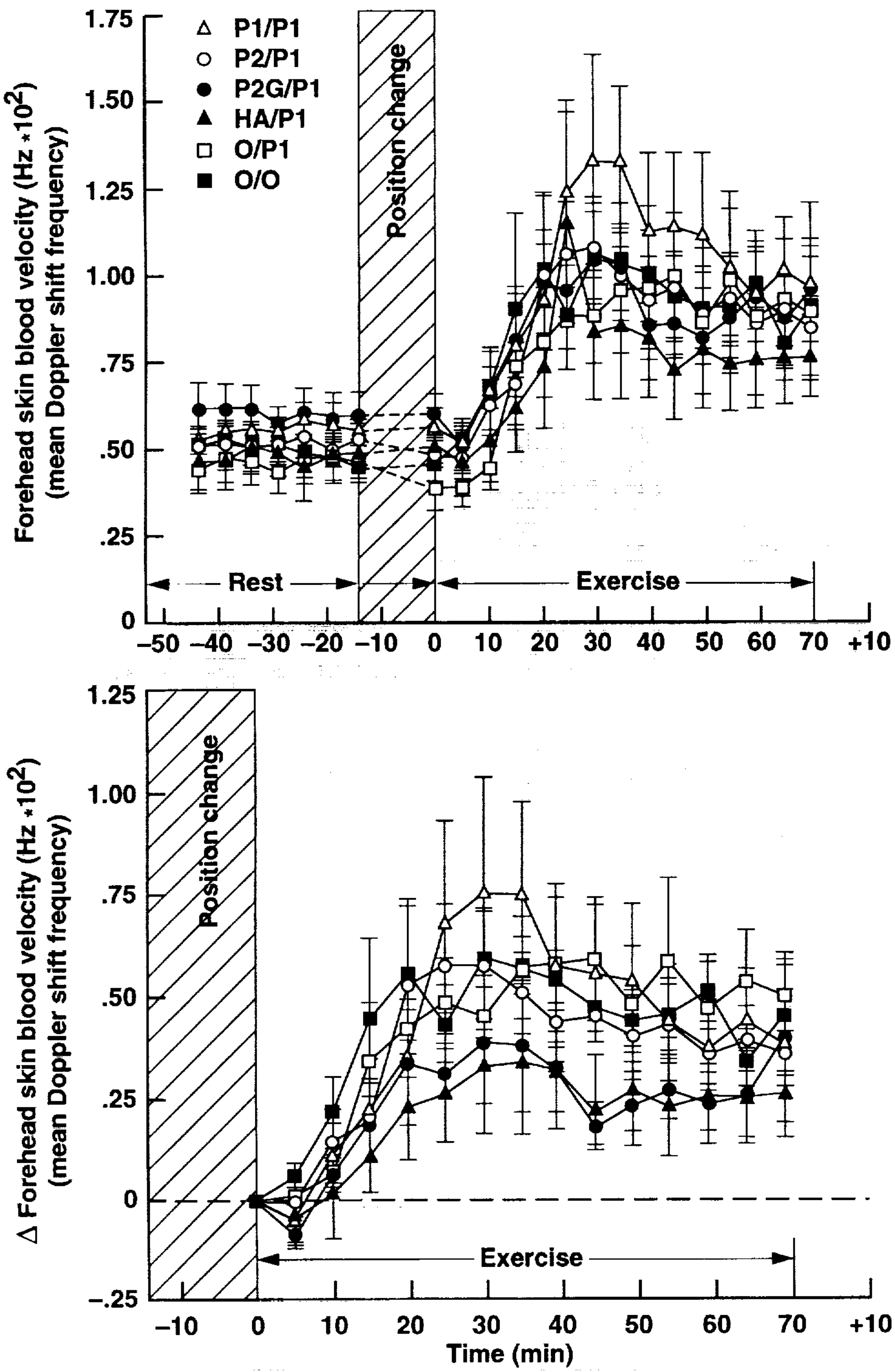

Figure 20. Mean (±SE) forehead skin blood velocity at rest and during exercise for the six treatments.

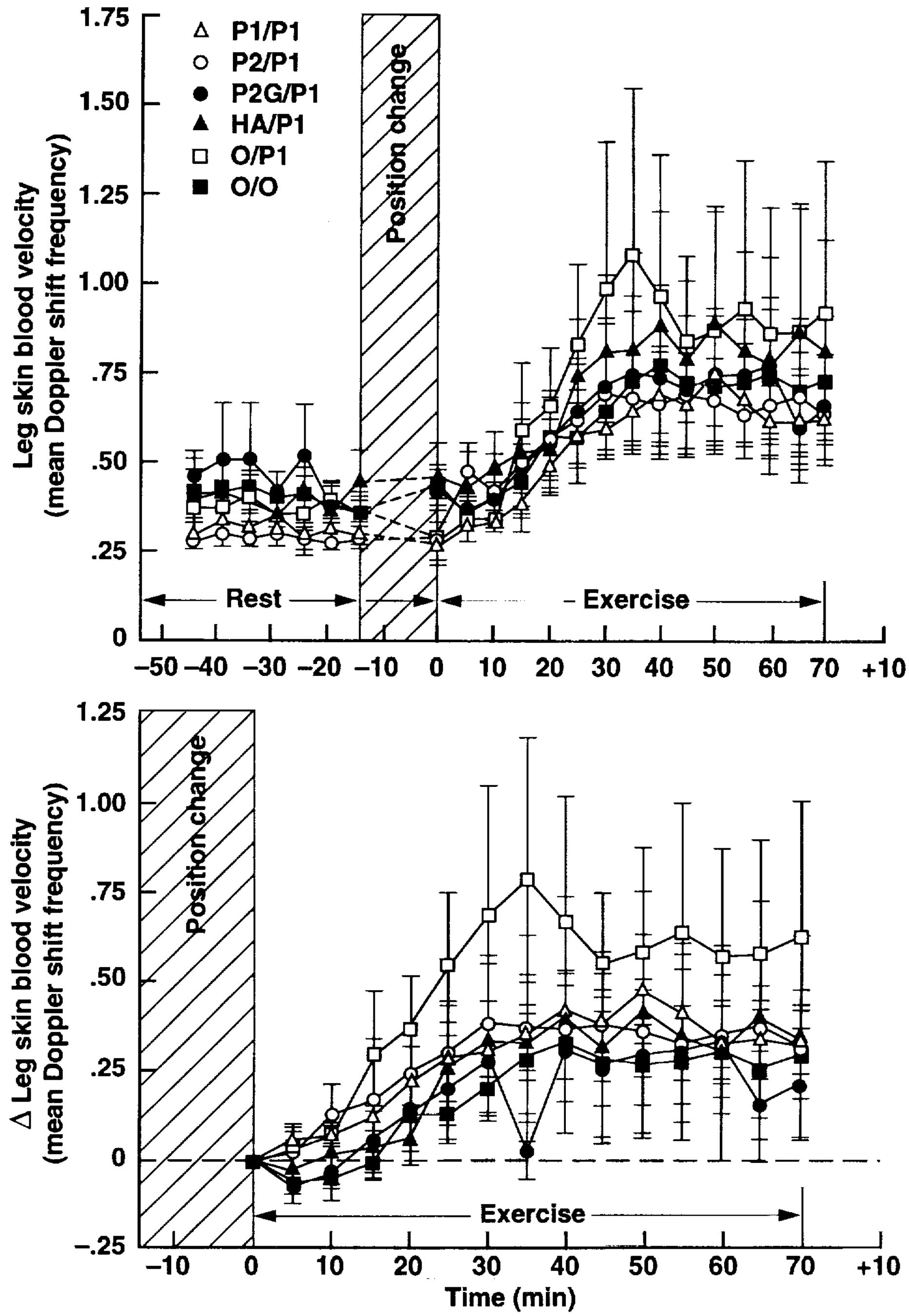

Figure 21. Mean (±SE) leg skin blood velocity at rest and during exercise for the six treatments.

Appendix 1. Mean metabolic data at rest and during exercise for the six treatments

		P1P1	P2P1	P2GP1	HAP1	0P1	00	Mean
				Rest phase (–45 min)				
$\dot{V}_{ESTPD}$	$\overline{X}$	11.42	11.33	10.92	11.81	10.05	10.17	10.95
	±SD	2.73	1.60	1.56	3.10	2.85	2.76	0.71
	±SE	1.11	0.65	0.64	1.26	1.16	1.13	0.29
R_E	$\overline{X}$	0.97	0.96	0.94	0.95	0.91	0.97	0.95
	±SD	0.06	0.02	0.07	0.05	0.06	0.09	0.02
	±SE	0.02	0.01	0.03	0.02	0.02	0.04	0.01
$\dot{V}O_2$ (lLmin)	$\overline{X}$	0.40	0.39	0.40	0.40	0.36	0.35	0.38
	±SD	0.08	0.05	0.03	0.08	0.07	0.08	0.02
	±SE	0.03	0.02	0.01	0.03	0.03	0.03	0.01
$\dot{V}O_2$ (mL/min · kg)	$\overline{X}$	5.6	5.4	5.8	5.8	4.6	4.6	5.3
	±SD	1.0	0.6	1.1	0.5	0.5	0.3	0.6
	±SE	0.4	0.2	0.4	0.2	0.2	0.1	0.2
				Exercise phase (35 min)				
$\dot{V}_{ESTPD}$	$\overline{X}$	48.45	48.74	50.64	49.54	51.81	48.60	49.63
	±SD	10.89	12.04	10.76	10.13	16.96	13.85	1.34
	±SE	4.45	4.92	4.39	4.13	6.92	5.65	0.55
R_E	$\overline{X}$	0.97	0.96	0.98	0.94	0.96	0.96	0.96
	±SD	0.02	0.02	0.07	0.07	0.03	0.02	0.01
	±SE	0.01	0.01	0.03	0.03	0.01	0.01	0.01
$\dot{V}O_2$ (L/min)	$\overline{X}$	2.01	2.09	2.08	2.12	2.03	2.02	2.06
	±SD	0.32	0.35	0.28	0.38	0.38	0.38	0.04
	±SE	0.13	0.14	0.11	0.15	0.15	0.15	0.02
$\dot{V}O_2$ (mL/min · kg)	$\overline{X}$	28.2	29.1	29.6	29.9	27.4	26.8	28.5
	±SD	4.4	6.1	3.7	4.2	3.9	3.8	1.2
	±SE	1.8	2.5	1.5	1.7	1.6	1.5	0.5
				Exercise phase (65 min)				
$\dot{V}_{ESTPD}$	$\overline{X}$	48.19	49.41	48.59	47.70	50.37	48.38	48.94
	±SD	11.51	10.79	9.53	8.43	13.78	10.51	0.93
	±SE	4.55	4.40	3.89	3.44	5.63	4.29	0.38
R_E	$\overline{X}$	0.94	0.95	0.96	0.94	0.94	0.94	0.95
	±SD	0.05	0.02	0.04	0.03	0.04	0.04	0.01
	±SE	0.02	0.01	0.02	0.01	0.02	0.02	0.01

Appendix 1. Concluded

$\dot{V}O_2$ (L/min)	$\overline{X}$	2.14	2.12	2.05	2.11	2.12	2.10	2.11
	±SD	0.38	0.32	0.29	0.30	0.38	0.35	0.03
	±SE	0.16	0.13	0.12	0.12	0.15	0.14	0.01
$\dot{V}O_2$ (mL/min · kg)	$\overline{X}$	29.9	29.4	29.1	29.6	28.6	27.9	29.1
	±SD	4.7	5.0	3.5	4.1	3.4	4.0	0.7
	±SE	1.9	2.0	1.4	1.7	1.4	1.6	0.3

Appendix 2. Mean plasma citrate concentration at rest and during exercise for the six treatments

		Rest phase		Exercise phase		
Time (min)		−105	0	10	30	70
Treatment						
	$\overline{X}$	1.7	2.3	2.4	1.9	2.4
P1P1	±SD	0.4	0.8	0.7	0.7	0.6
	±SE	0.2	0.3	0.3	0.3	0.2
	$\overline{X}$	1.8	2.5	2.7	2.4	2.6
P2P1	±SD	0.5	0.7	0.8	0.8	0.7
	±SE	0.2	0.3	0.3	0.3	0.3
	$\overline{X}$	1.8	2.2	2.4	2.5	2.6
P2GP1	±SD	0.8	0.6	0.7	0.7	0.6
	±SE	0.3	0.3	0.3	0.3	0.3
	$\overline{X}$	1.8	3.4	2.9	2.7	2.5
HAP1	±SD	0.5	0.6	0.6	0.5	0.6
	±SE	0.2	0.3	0.3	0.2	0.2
	$\overline{X}$	2.2	1.8	2.0	1.8	2.3
0P1	±SD	0.6	0.7	0.5	0.7	0.7
	±SE	0.3	0.3	0.2	0.3	0.3
	$\overline{X}$	1.9	1.7	1.9	1.9	2.1
00	±SD	0.7	0.5	0.9	0.6	0.5
	±SE	0.3	0.2	0.4	0.2	0.2

Normal range = 1.7 – 3.0 mg/dL.

Appendix 3. Mean hemoglobin concentration and hematocrit at rest and during exercise for the six treatments

		Rest phase			Exercise phase			
				Hemoglobin				
Time (min)		−105	−30	−25	0	10	30	70
Treatment								
	$\overline{X}$	16.3	16.4	16.3	16.4	17.0	17.0	16.5
P1P1	±SD	0.5	1.0	0.6	0.8	0.7	0.6	0.6
	±SE	0.2	0.4	0.2	0.3	0.3	0.3	0.2
	$\overline{X}$	16.6	16.4	16.4	16.6	17.0	17.1	17.0
P2P1	±SD	0.4	0.7	0.6	0.6	0.6	0.4	0.7
	±SE	0.2	0.3	0.2	0.3	0.2	0.2	0.3
	$\overline{X}$	16.6	16.6	16.5	16.4	17.0	17.0	16.8
P2GP1	±SD	0.4	0.5	0.4	0.6	0.8	0.6	0.5
	±SE	0.2	0.2	0.2	0.3	0.3	0.2	0.2
	$\overline{X}$	16.6	16.1	16.0	16.1	16.6	16.4	16.4
HAP1	±SD	0.5	0.4	0.5	0.8	0.4	0.5	0.6
	±SE	0.2	0.1	0.2	0.3	0.2	0.2	0.3
	$\overline{X}$	17.4	16.8	16.9	17.3	18.0	17.7	17.4
0P1	±SD	1.1	1.1	1.1	1.0	1.2	1.4	1.4
	±SE	0.4	0.4	0.4	0.4	0.5	0.6	0.6
	$\overline{X}$	16.6	16.6	16.4	16.8	17.5	17.3	17.2
00	±SD	0.3	0.7	0.5	0.9	0.5	0.8	0.9
	±SE	0.1	0.3	0.2	0.4	0.2	0.3	0.4

Normal range = 13.6 – 17.2 g/dL.

				Hematocrit				
Time (min)		−105	−30	−25	0	10	30	70
Treatment								
	$\overline{X}$	45.3	45.9	45.6	45.8	47.4	46.8	46.0
P1P1	±SD	1.5	1.8	1.7	2.5	2.2	1.7	2.1
	±SE	0.6	0.7	0.7	1.0	0.9	0.7	0.9
	$\overline{X}$	46.7	46.2	46.0	46.5	47.8	47.5	46.5
P2P1	±SD	2.3	1.9	1.9	1.9	1.7	1.2	1.0
	±SE	0.9	0.8	0.8	0.8	0.7	0.5	0.4
	$\overline{X}$	46.8	46.6	46.2	46.7	48.1	47.3	47.0
P2GP1	±SD	1.7	1.8	1.4	1.9	1.9	2.0	1.9
	±SE	0.7	0.7	0.6	0.8	0.8	0.8	0.8
	$\overline{X}$	46.4	45.3	44.9	45.6	46.1	45.4	45.2
HAP1	±SD	1.2	1.0	1.4	2.6	1.9	2.0	2.2
	±SE	0.5	0.4	0.6	1.1	0.8	0.8	0.9
	$\overline{X}$	48.4	47.0	47.2	48.4	50.1	49.1	48.1
0P1	±SD	2.1	2.2	1.9	2.0	2.4	2.9	2.8
	±SE	0.8	0.9	0.8	0.8	1.0	1.2	1.1
	$\overline{X}$	46.6	46.1	46.4	47.1	49.0	47.9	47.3
00	±SD	0.8	1.4	1.5	1.8	1.6	1.7	2.1
	±SE	0.3	0.6	0.6	0.7	0.6	0.7	0.9

Normal range = 39% – 49%.

Appendix 4. Mean red blood cell and white blood cell (leukocyte) concentrations at rest and during exercise for the six treatments

		Rest phase				Exercise phase		
				Red cells				
Time (min)		−105	−30	−25	0	10	30	70
Treatment								
	$\overline{X}$	5.33	5.26	5.23	5.24	5.48	5.43	5.38
P1P1	±SD	0.30	0.25	0.24	0.24	0.26	0.20	0.28
	±SE	0.12	0.10	0.10	0.10	0.11	0.08	0.11
	$\overline{X}$	5.36	5.30	5.27	5.33	5.50	5.54	5.46
P2P1	±SD	0.31	0.20	0.25	0.22	0.20	0.20	0.20
	±SE	0.13	0.08	0.10	0.09	0.08	0.08	0.08
	$\overline{X}$	5.34	5.32	5.28	5.33	5.53	5.49	5.47
P2GP1	±SD	0.35	0.30	0.28	0.33	0.35	0.33	0.32
	±SE	0.14	0.12	0.11	0.14	0.14	0.14	0.13
	$\overline{X}$	5.32	5.18	5.14	5.23	5.32	5.30	5.27
HAP1	±SD	0.27	0.18	0.22	0.31	0.31	0.26	0.27
	±SE	0.11	0.07	0.09	0.13	0.13	0.10	0.11
	$\overline{X}$	5.62	5.48	5.43	5.55	5.78	5.75	5.66
0P1	±SD	0.27	0.30	0.23	0.23	0.30	0.38	0.38
	±SE	0.11	0.12	0.09	0.09	0.12	0.16	0.16
	$\overline{X}$	5.39	5.34	5.36	5.42	5.71	5.64	5.55
00	±SD	0.15	0.17	0.19	0.15	0.21	0.19	0.28
	±SE	0.06	0.07	0.08	0.06	0.09	0.08	0.11

Normal range = 4.3–5.9 × $10^6/mm^3$.

				White cells				
Time (min)		−105	−30	−25	0	10	30	70
Treatment								
	$\overline{X}$	5.2	4.9	4.9	5.5	7.2	7.4	7.1
P1P1	±SD	1.9	1.8	1.7	1.3	2.0	2.4	2.7
	±SE	0.8	0.8	0.7	0.5	0.8	1.0	1.1
	$\overline{X}$	4.6	4.3	4.7	5.2	6.4	7.0	7.1
P2P1	±SD	1.4	1.2	0.7	0.6	1.8	1.9	2.0
	±SE	0.6	0.5	0.3	0.3	0.7	0.8	0.8
	$\overline{X}$	4.7	4.5	4.5	5.2	6.8	7.4	7.1
P2GP1	±SD	0.9	1.0	1.1	1.4	1.8	2.2	2.1
	±SE	0.4	0.4	0.4	0.6	0.7	0.9	0.9
	$\overline{X}$	4.9	4.6	4.7	5.5	6.2	6.6	6.6
HAP1	±SD	1.0	0.9	0.8	1.8	1.3	1.5	1.5
	±SE	0.4	0.4	0.3	0.8	0.5	0.6	0.6
	$\overline{X}$	5.6	5.2	5.1	5.5	7.6	8.0	7.9
0P1	±SD	1.0	0.6	0.7	0.6	1.0	1.2	1.4
	±SE	0.4	0.3	0.3	0.3	0.4	0.5	0.6
	$\overline{X}$	4.6	4.7	4.6	5.0	7.0	7.0	7.5
00	±SD	1.1	1.3	1.3	1.4	2.1	2.4	3.4
	±SE	0.5	0.5	0.5	0.6	0.8	1.0	1.4

Normal range = 3.2–9.8 × $10^3/mm^3$.

Appendix 5. Mean platelet (thrombocyte) concentration at rest and during exercise for the six treatments

		Rest phase				Exercise phase		
Time (min)		–105	–30	–25	0	10	30	70
Treatment								
	$\overline{X}$	210	210	204	206	226	250	260
P1P1	±SD	33	44	38	34	43	49	60
	±SE	13	18	16	14	18	20	24
	$\overline{X}$	210	212	206	217	240	259	246
P2P1	±SD	50	58	53	64	59	54	88
	±SE	20	24	22	26	24	22	36
	$\overline{X}$	211	216	212	216	244	248	270
P2GP1	±SD	46	47	50	47	47	57	60
	±SE	19	19	20	19	19	23	25
	$\overline{X}$	218	210	209	204	226	224	240
HAP1	±SD	47	42	40	23	50	60	51
	±SE	19	17	16	10	20	25	21
	$\overline{X}$	215	225	220	220	239	248	279
0P1	±SD	32	33	48	53	47	54	40
	±SE	13	14	19	22	19	22	16
	$\overline{X}$	202	206	198	206	221	232	240
00	±SD	43	41	43	41	33	35	38
	±SE	18	17	18	17	13	14	16

Normal range = 150 – 450 × $10^3/mm^3$.

Appendix 6. Individual resting hematocrit and plasma and blood volumes for the six treatments

Subject	Date (treatment)	Corrected absorbance pre-dye/post-dye	Dye injected, mL	Hct, %	Plasma volume, mL	Blood volume, mL
CAL	8/19/93	0.005				
	(00)	0.037	2.6123	46.3	3240	5598
	8/26/93	0.011				
	(P1P1)	0.051	2.5329	45.4	2536	4321
	9/2/93	0.008				
	(0P1)	0.050	2.7194	45.5	2562	4372
	9/16/93	0.010				
	(P2GP1)	0.052	2.5604	48.6	2471	4430
	9/23/93	0.020				
	(HAP1)	0.050	2.6036	46.0	3539	6087
	9/27/93	0.024				
	(P2P1)	0.052	2.4783	46.8	3609	6286
	$\overline{X}$		2.5845	46.4	2993	5182
	±SD		0.0823	1.2	530	914
	±SE		0.0336	0.5	216	373
DUW	8/18/93	0.005				
	(0P1)	0.031	2.6860	48.6	4112	7373
	8/25/93	0.009				
	(00)	0.038	2.5103	46.4	3467	6000
	9/1/93	0.008				
	(HAP1)	0.034	2.5974	46.7	3880	6747
	9/8/93	0.008				
	(P1P1)	0.034	2.5701	46.8	3911	6812
	9/15/93	0.016				
	(P2P1)	0.040	2.5502	45.7	4178	7153
	9/22/93	0.014				
	(P2GP1)	0.037	2.5804	47.0	4575	7994
	$\overline{X}$		2.5824	46.9	4020	7013
	±SD		0.0589	1.0	368	671
	±SE		0.0240	0.4	150	274
GUF	8/19/93	0.004				
	(00)	0.044	2.5716	49.2	2551	4620
	8/26/93	0.013				
	(P1P1)	0.062	2.6174	48.5	2139	3829
	9/2/93	0.015				
	(0P1)	0.055	2.6029	45.8	2527	4333
	9/9/93	0.015				
	(P2P1)	0.065	2.6039	48.9	2060	3712
	9/16/93	0.031				
	(P2GP1)	0.062	2.4945	46.4	3164	5476
	9/23/93	0.023				
	(HAP1)	0.065	2.5431	45.8	2454	4208

Appendix 6. Continued

	$\overline{X}$		2.5732	47.4	2482	4363
	±SD		0.0475	1.6	392	639
	±SE		0.0194	0.6	160	261
PAU	8/18/93	0.005				
	(0P1)	0.041	2.6221	50.2	2899	5338
	8/25/93	0.010				
	(00)	0.046	2.5099	44.7	2792	4707
	9/1/93	0.010				
	(HAP1)	0.044	2.5532	43.6	2916	4834
	9/8/93	0.012				
	(P1P1)	0.047	2.6284	44.5	2971	4993
	9/15/93	0.020				
	(P2P1)	0.050	2.5706	43.8	3369	5602
	9/22/93	0.014				
	(P2GP1)	0.054	2.5799	45.0	2614	4427
	$\overline{X}$		2.5744	45.3	2927	4984
	±SD		0.0443	2.5	251	428
	±SE		0.0181	1.0	102	175
PED	8/17/93	0.003				
	(P2P1)	0.034	2.5038	46.7	3215	5591
	8/24/93	0.012				
	(0P1)	0.044	2.5538	47.5	3167	5578
	8/31/93	0.013				
	(P2GP1)	0.045	2.6018	45.1	3158	5355
	9/7/93	0.015				
	(00)	0.055	2.5890	46.4	2561	4432
	9/14/93	0.026				
	(HAP1)	0.052	2.5791	43.5	3900	6456
	9/21/93	0.019				
	(P1P1)	0.058	2.6069	43.7	2709	4498
	$\overline{X}$		2.5724	45.5	3118	5318
	±SD		0.0385	1.6	470	761
	±SE		0.0157	0.7	192	311
REA	8/16/93	0.004				
	(P1P1)	0.041	2.5370	44.9	2729	4615
	8/24/93	0.009				
	(HAP1)	0.040	2.5617	45.6	3279	5605
	8/30/93	0.009				
	(P2P1)	0.040	2.5069	44.3	3140	5262
	9/13/93	0.015				
	(P2GP1)	0.044	2.5124	45.4	3406	5805
	9/20/93	0.012				
	(0P1)	0.050	2.5330	45.7	2702	4626
	9/27/93	0.022				
	(00)	0.046	2.4971	45.6	4243	7252

Appendix 6. Concluded

$\overline{X}$	2.5247	45.2	3250	5528
±SD	0.0237	0.6	565	977
±SE	0.0097	0.2	231	399

From –35 to –25 min.

Appendix 7. Individual blood and plasma variables at rest and during exercise for the six treatments

Time, min	Treatment	White blood cells 3.4–10.0 × 1000	Red blood cells 4.4–5.9 M/UL	Hemoglobin 13.5–17.5 gm/dL	Hematocrit 41–53%	Platelets 130–400 × 1000	Sodium, plasma 135–148 mEq/L	Potassium, plasma 3.6–5.0 mEq/L	Glucose, plasma 64–115 mg/dL	Glycerol, plasma 3–17 mg/dL[b]	Citrate, plasma 1.7–3.0 mg/dL
						Subject CAL					
	P1P1	5	5.71	15.9	48.8	191	146.9	4.5	90	4	1.6[a]
	P2P1	2.4[a]	5.81	16.4	49.3	160	146.4	4.5	144[a]	16	2.7
−105	P2GP1	5.1	5.83	16.6	49.1	195	146.9	4.2	134[a]	6	3.3[a]
	HAP1	5.1	5.7	16.2	49.1	167	146.5	4.8	109	8	2.5
	0P1	6.5	5.75	16.4	49.1	210	147.8	4.3	105	12	2.9
	00	4.6	5.59	15.7	48	162	166.1[a]	4.3	91	8	1.7
	P1P1	4.5	5.51	15.6	47	175	146.6	4.3	78	3	–
	P2P1	2.1[a]	5.54	15.8	47	162	147.2	4.2	132[a]	9	–
−35	P2GP1	4.9	5.78	16.5	49.3	193	146.9	4	135[a]	105[a]	–
	HAP1	5.8	5.44	15.4	46.5	172	146.1	4.7	74	6	–
	0P1	5.4	5.29	15.2	45.1	202	145.6	4.1	132[a]	8	–
	00	4.8	5.41	15.4	46.4	167	146.8	4.4	74	5	–
	P1P1	4.6	5.35	15.1	45.8	165	146.8	4.1	74	8	–
	P2P1	2.2[a]	5.61	15.8	47.8	154	147	4.1	128[a]	17	–
−25	P2GP1	5	5.7	16.3	48.3	177	147.6	3.8	99	102[a]	–
	HAP1	5.6	5.44	15.4	46.2	165	145.7	4.7	75	9	–
	0P1	5.5	5.35	15.2	45.7	184	146	4	105	12	–
	00	5.5	5.54	15.7	47.9	163	147	4.3	61[a]	21[a]	–
	P1P1	4.9	5.47	15.5	46.7	180	146.5	4	52[a]	9	2.8
	P2P1	2.5[a]	5.66	15.7	48.1	165	146.7	4.5	122[a]	9	3.1[a]
0	P2GP1	6.4	5.9	16.8	50.6	200	148.9[a]	4	54[a]	96[a]	2.9
	HAP1	6.3	5.67	16	48.5	173	145.9	4.7	67	8	3.9[a]
	0P1	5.7	5.56	15.9	47.4	198	148.3[a]	3.8	72	11	2.2
	00	5.2	5.37	15.2	45.7	163	155.1[a]	4.5	77	7	2.2

Appendix 7. Continued

	P1P1	6.9	5.71	16.3	49.2	197	147.6	4.5	44[a]	8	2.9
	P2P1	3.4	5.82	16.5	49.6	181	148.9[a]	4.4	69	13	3.7[a]
10	P2GP1	8.5	6.11[a]	17.5	52.5	227	149.8[a]	4.5	48[a]	96[a]	2.8
	HAP1	7.9	5.84	16.7	50.2	174	147.1	5.1[a]	80	8	3.5[a]
	0P1	7.8	5.76	16.5	49.3	233	148.9[a]	4.1	64	29[a]	2.5
	00	7.5	5.96[a]	16.9	51	184	147	5.2[a]	70	8	1.3[a]
	P1P1	7.5	5.53	15.7	47.8	219	147.2	5.1[a]	69	7	0.4[a]
	P2P1	3.8	5.89	16.4	50.2	205	149.1[a]	4.9	74	11	3.4[a]
30	P2GP1	9.2	5.98[a]	17.1	50.9	247	149.9[a]	4.6	53[a]	95[a]	3.2[a]
	HAP1	7.2	5.61	15.9	48	150	141.2	4.9	99	6	3.4[a]
	0P1	8.5	5.74	16.3	48.8	248	149.1[a]	4.7	83	30[a]	2.2
	00	7.1	5.64	16	47.7	199	148.1	5	65	7	2.6
	P1P1	8.4	5.54	15.8	47.5	238	147.6	5.1[a]	84	11	3
	P2P1	3.8	5.8	16.4	49.4	210	148	4.9	102	8	3.5[a]
70	P2GP1	9.5	5.91[a]	16.9	50.7	253	149.3	5	75	64[a]	3.2[a]
	HAP1	7.5	5.63	15.9	48.4	175	146.5	5.1[a]	88	8	3.6[a]
	0P1	8.8	5.63	16	47.8	270	147.8	5	93	15	2.8
	00	9.6	5.46	15.6	46.6	216	159.8[a]	5	91	8	2.5
					Subject DUW						
	P1P1	5.5	5.3	16.5	48.2	164	145.2	4.8	105	4	2.2
	P2P1	5.3	5.01	15.6	46	161	144.8	4.9	129[a]	4	1.6[a]
−105	P2GP1	5.5	5.16	15.9	47.5	169	144.2	4.6	129[a]	5	1.6[a]
	HAP1	5.3	5.25	16.3	48	178	144.3	4.7	124[a]	6	2.2
	0P1	6.6	5.71	17.2	52.7	195	146.1	4.6	124[a]	8	2.2
	00	6.2	5.36	16.2	48.9	171	143.6	5.3[a]	134[a]	11	2.3
	P1P1	6.7	5.25	16.2	48.2	158	146.1	4.5	72	4	–
	P2P1	5.7	5.18	16.1	47.6	164	146.2	4.5	79	4	–
−35	P2GP1	5.5	5.16	16	47.5	173	145.9	4.1	91	5	–
	HAP1	4.8	5.15	16.2	47.4	176	145.5	4.6	65	3	–
	0P1	6	5.7	17.1	52.9	190	145.6	4.7	80	6	–
	00	6.1	5.28	16.1	48.6	166	145.7	4.9	99	5	–

Appendix 7. Continued

	P1P1	6.5	5.13	16.1	46.9	168	145.6	4.7	71	7	–
	P2P1	5.6	4.99	15.7	45.8	161	146.2	4.1	87	5	–
–25	P2GP1	5.9	5.21	16	48.2	163	145.9	4.1	84	6	–
	HAP1	5	5.14	16	47.4	175	145.8	4.3	49[a]	8	–
	0P1	5.7	5.51	17	50.5	181	145.2	4.4	80	8	–
	00	6	5.26	16.3	48.3	159	145.2	4.5	88	12	–
	P1P1	6.9	5.24	16.2	48.1	169	145.9	4.7	60[a]	6	2.1
	P2P1	5.8	5.2	16.3	47.7	163	146.9	4.2	59[a]	4	2.4
0	P2GP1	7	5.31	16.3	48.8	172	146.6	4.3	52[a]	6	2.3
	HAP1	8.6	5.42	17	49.4	184	145.1	4.9	49[a]	8	3.4[a]
	0P1	6	5.67	17.6[a]	52.6	180	146.3	5.1	79	7	1.1[a]
	00	6.9	5.43	16.7	50.1	175	145.3	4.5	70	12	1.9
	P1P1	9.9	5.49	17.1	50.4	194	147.9	5.1[a]	53[a]	5	2.3
	P2P1	8.7	5.44	16.9	49.6	187	146.7	5.2[a]	64	5	3.2[a]
10	P2GP1	9.3	5.55	17.2	51	177	147.8	4.9	54[a]	6	2.9
	HAP1	5.7	5.14	16.1	47	181	145.9	5.6[a]	61[a]	8	Missing
	0P1	8.6	6.02[a]	18.4[a]	55.8[a]	187	147.6	5.3[a]	63[a]	9	1.5[a]
	00	10.2[a]	5.77	17.9[a]	52.8	197	147.4	5.3[a]	67	17	1.7
	P1P1	11[a]	5.63	17.3	51.9	217	147.7	5.4[a]	67	6	2.2
	P2P1	9.4	5.53	17	50.8	200	147.9	5.3[a]	71	6	2.4
30	P2GP1	10.6[a]	5.52	17.2	50.8	200	147.6	5.2[a]	83	9	2.9
	HAP1	8.7	5.43	17	49.9	197	146.9	5.4[a]	97	11	2.7
	0P1	9.5	6.01[a]	18.6[a]	56[a]	196	148.1[a]	5.7[a]	92	13	1.1[a]
	00	11.0[a]	5.89	18.2[a]	53.7[a]	207	148.1[a]	5.5[a]	65	23[a]	2.1
	P1P1	10.8[a]	5.55	17.3	50.9	226	147.4	5.5[a]	101	9	2.4
	P2P1	9.4	5.51	17	50.5	195	148.5	5.5[a]	101	6	2.8
70	P2GP1	9.7	5.61	17.3	51.2	202	147.6	5.4[a]	96	11	2.5
	HAP1	8.8	5.47	17	50.3	223	147.7	5.5[a]	96	11	2.3
	0P1	9.2	6.03[a]	18.7[a]	55.8[a]	220	146.2	5.7	104	11	2.7
	00	13.0[a]	5.92[a]	18.4[a]	55.0[a]	235	148.4	5.5[a]	73	32[a]	2.8

Appendix 7. Continued

						Subject GUF					
	P1P1	8.7	5.06	15.3	45.1	229	145.7	4.3	107	6	1.3[a]
	P2P1	5.2	5.57	16.9	49.4	226	146.4	3.9	111	6	1.7
−105	P2GP1	5.3	5.28	16	47.6	209	145.9	4.5	113	7	0.8[a]
	HAP1	5.6	5.04	15.3	45.4	236	145.8	4.2	109	6	1.9
	0P1	Clotted	Clotted	Clotted	Clotted	Clotted	146.2	4	132[a]	10	2.2
	00	5.2	5.25	15.7	46.9	215	145.4	4.1	113	9	2.7
	P1P1	7.5	5.42	16.4	48.6	222	146.7	4.1	58[a]	7	–
	P2P1	4.4	5.51	16.8	49.3	217	148.3[a]	4	62[a]	6	–
−35	P2GP1	5	5.37	16.4	48.1	213	148.1[a]	4.7	80	74[a]	–
	HAP1	4.9	5.06	15.5	45.5	196	147.9	4.7	66	5	–
	0P1	4.4	5.07	15.5	45.1	236	146.9	4.3	95	7	–
	00	5.8	5.53	16.6	49	249	145.7	4.4	74	8	–
	P1P1	7.2	5.46	16.5	49	229	147.8	3.8	59[a]	11	–
	P2P1	4.3	5.48	16.7	49.1	215	148.4[a]	3.8	50[a]	8	–
−25	P2GP1	5	5.2	15.9	47.1	214	150.3[a]	5.9[a]	111	140[a]	–
	HAP1	5.1	5.1	15.6	46	211	149[a]	5.2[a]	76	8	–
	0P1	3.9	5.18	15.8	46.4	229	147	4.1	82	13	–
	00	4.4	5.55	16.6	49.4	203	147.7	4.2	66	23[a]	–
	P1P1	7.2	5.54	16.7	49.6	219	148.1[a]	3.8	48[a]	16	3.5[a]
	P2P1	4.8	5.51	16.7	49.4	219	148.8[a]	3.5	52[a]	9	2.3
0	P2GP1	5.2	5.07	15.6	45.6	240	146.4	3.6	57[a]	122[a]	1.8
	HAP1	5.3	5.14	15.6	46.4	218	148.4[a]	5.1[a]	68	8	3.7[a]
	0P1	4.7	5.22	16.1	46.9	232	148.3[a]	4	64	13	2.4
	00	4.8	5.65	17	50	211	144.6	4.6	57[a]	20[a]	2
	P1P1	9.2	5.69	17.1	50.9	257	149[a]	4.5	42[a]	20[a]	3.4[a]
	P2P1	5.8	5.64	17	50.3	246	149.2[a]	4.1	35[a]	9	2.7
10	P2GP1	6.4	5.23	16	47.1	244	147.9	4.4	34[a]	121[a]	2.2
	HAP1	6.4	5.13	15.6	45.9	240	148.9[a]	4.3	51[a]	10	3.7[a]
	0P1	5.9	5.48	16.7	48.9	256	149.8[a]	4.7	66	13	2.4
	00	6.1	5.86	17.1	52.1	226	144.9	4.5	70	13	3.7[a]

Appendix 7. Continued

	P1P1	9.1	5.54	16.7	49.4	267	148.6[a]	5.3[a]	64	12	1.8
	P2P1	6.4	5.44	16.5	48.7	264	148.5[a]	5.1[a]	66	8	2
30	P2GP1	6.6	5.19	16	46.5	234	148.1[a]	5	58[a]	139[a]	1.9
	HAP1	6.3	4.97	15.3	44.6	235	147.2	5.3[a]	82	9	3
	0P1	6	5.37	16.3	48.2	268	148.9[a]	5	77	15	1.6[a]
	00	5.9	5.59	16.8	49.8	236	144.9	4.9	77	14	2.6
	P1P1	8.7	5.46	16.5	48.9	281	149.3[a]	5.2[a]	72	15	1.9
	P2P1	6.3	5.46	16.6	49.2	273	149.9[a]	5.3[a]	75	10	2.5
70	P2GP1	6	5.2	15.8	46.7	267	148.7[a]	5.1[a]	70	120[a]	1.9
	HAP1	6.1	4.98	15.2	44.7	241	148.3[a]	5.3[a]	94	10	2.6
	0P1	5.5	5.26	16.2	46.5	294	148.1[a]	4.9	98	14	1.8
	00	4.9	5.59	16.6	49.8	184	154.4[a]	5.3[a]	88	17	2.2
					Subject PAU						
	P1P1	4.4	5.16	15.7	45.7	232	145.9	4.5	138[a]	4	1.8
	P2P1	4.1	5.08	15.5	45	230	144.6	4.3	146[a]	4	1.9
−105	P2GP1	4	5.3	16.1	47	230	144.7	4.3	156[a]	5	2.2
	HAP1	4.4	5.25	16	46.6	238	143.9	4.4	150[a]	8	2
	0P1	4.8	5.95[a]	17.7[a]	52.7	228	145.3	4.5	114[a]	7	2.7
	00	4.6	5.37	16	47.8	213	141.2	4.1	146[a]	5	1.6[a]
	P1P1	4.4	5.05	15.4	44.9	218	145.7	4.3	131[a]	4	–
	P2P1	3.8	5.06	15.3	44.6	225	146.6	4.5	107	4	–
−35	P2GP1	4.1	5.17	15.8	46.1	223	146.3	4.4	130[a]	80[a]	–
	HAP1	4.2	5.1	15.5	45.1	232	145.5	4.5	76	6	–
	0P1	4.5	5.91[a]	17.5	53.1[a]	225	144.6	5.1[a]	108	5	–
	00	4.8	5.18	15.6	46.2	216	141.8	4.4	105	5	–
	P1P1	4.4	5.14	15.6	45.7	218	145.6	4.4	123[a]	23[a]	–
	P2P1	3.9	5.02	15.3	44.5	217	146.7	4.4	102	6	–
−25	P2GP1	4	5.15	15.7	45.7	230	146.7	4.3	133[a]	105[a]	–
	HAP1	4.4	5.07	15.4	44.7	236	146.3	4.6	73	8	–
	0P1	4.8	5.83	17.6[a]	51.8	216	144.8	4.7	112	7	–
	00	5.1	5.21	15.5	46.4	221	143.3	4.4	98	17	–

Appendix 7. Continued

	P1P1	4.5	5.03	15.3	44.9	221	145.9	4.5	113	6	2.3
	P2P1	4.4	5.11	15.6	45.1	225	146.4	4.4	98	5	2.9
0	P2GP1	4.6	5.23	15.7	46.5	203	146.4	4.4	122[a]	137[a]	2.6
	HAP1	4.3	4.95	15.2	43.5	226	145.6	4.9	74	8	4
	0P1	5	5.89	17.8[a]	52.3	219	144.7	4.6	113	8	2.1
	00	5.3	5.23	15.8	47.1	231	142.9	4.7	90	12	1.5[a]
	P1P1	6.6	5.32	16.1	47.4	253	148	5.1[a]	84	6	2.3
	P2P1	6.1	5.31	16.2	47.2	262	148.7	5.4[a]	78	14	2.9
10	P2GP1	6.2	5.46	16.7	48.8	255	148.2	4.9	119[a]	157[a]	2.4
	HAP1	6.4	5.3	16.2	47.2	252	147.1	5.5[a]	106	9	3
	0P1	8.3	6.26[a]	18.7[a]	56[a]	248	147.6	5.6[a]	90	9	2.3
	00	7.4	5.54	16.8	50.1	251	146.3	5.3[a]	92	18[a]	1.5[a]
	P1P1	7.1	5.39	16.3	47.8	290	147.7	5.1[a]	84	10	2.6
	P2P1	7	5.38	16.3	48.2	290	148.4	5.2[a]	89	11	3
30	P2GP1	7.4	5.49	16.7	49	265	148.8	5.2[a]	97	139[a]	3
	HAP1	6.6	5.28	16	47	264	147.4	5.9[a]	111	12	2.9
	0P1	8.9	6.37[a]	19.2[a]	57.1[a]	289	147.9	5.8[a]	102	15	2.9
	00	7.7	5.66	17	50.9	257	147.3	5.4[a]	78	17	1.8
	P1P1	6.4	5.25	15.8	46.8	284	146.4	5.3[a]	114	7	2.9
	P2P1	6.7	5.28	16.1	46.9	287	147.5	5.4[a]	107	6	3.1[a]
70	P2GP1	6.7	5.48	16.5	48.7	317	147.4	5.2[a]	123[a]	80[a]	3.5[a]
	HAP1	6.4	5.21	15.8	46.1	287	146.3	5.6[a]	114	12	2.8
	0P1	8.9	6.21[a]	18.2[a]	55.4[a]	335	148.9	6.1[a]	142[a]	21[a]	3.3[a]
	00	7.7	5.57	16.8	50	286	148	5.6[a]	84	27[a]	2.1
						Subject PED					
	P1P1	4.5	5.06	15.6	45.7	191	147.4	4.6	115	5	1.9
	P2P1	6.4	5.23	16.2	47.5	191	143.3	3.8	138[a]	7	2
−105	P2GP1	4.9	4.85	15.1	44.1	173	146.4	4.2	121[a]	3	1.8
	HAP1	6	5.09	15.9	46.3	196	146.3	4.5	114	5	1.5[a]
	0P1	6.2	5.16	15.9	47	174	144.8	4.2	132[a]	8	2.4
	00	4.6	5.23	16.2	47.5	175	145.8	4.4	123[a]	5	2.4

Appendix 7. Continued

	P1P1	4	4.88	15.3	44.4	200	148.1	4.9	104	4	–
	P2P1	5.1	5.17	16.1	47	188	147.2	3.5	124[a]	6	–
–35	P2GP1	4.6	4.93	15.3	45.2	189	146.9	4	85	218[a]	–
	HAP1	4.8	4.96	15.4	45.8	201	148	4.1	61[a]	3	–
	0P1	5.7	5.37	16.4	48.9	214	147.2	4.5	79	6	–
	00	4.4	5.13	16	47.1	180	147	4.5	87	5	–
	P1P1	4.1	4.84	15	44.1	183	148.3	4.4	115	5	–
	P2P1	5.2	5.19	16.1	47.4	188	146.9	3.5	122[a]	13	–
–25	P2GP1	4.5	4.93	15.5	45.6	188	148.2	3.9	71	262[a]	–
	HAP1	4.8	4.8	15	44.1	193	147.1	4	62[a]	5	–
	0P1	5.7	5.25	16.3	47.9	198	146.5	4.5	74	9	–
	00	4.5	5.1	16	47	171	146.9	4.5	67	14	–
	P1P1	3.9	4.92	15.4	44.6	186	147.7	4.2	52[a]	6	2.3
	P2P1	5.9	5.17	16.3	47.1	194	146.5	3.5	106	8	2.9
0	P2GP1	5	4.98	15.5	45.6	183	148	4	68	326[a]	2.7
	HAP1	5	4.83	15.2	44.6	194	146.8	4	44[a]	5	3.3
	0P1	6.4	5.52	16.9	50.4	175	148.3	4.6	54[a]	9	2.4
	00	5.3	5.31	16.5	48.5	182	147.2	4.7	56[a]	8	2
	P1P1	5.6	5.04	15.8	45.8	175	147.9	4.8	56[a]	6	2.1
	P2P1	7.6	5.31	16.5	48.7	223	147.2	4.2	79	11	2.6
10	P2GP1	6.3	5.14	16	47.1	240	148.9	4.4	59[a]	303[a]	2.8
	HAP1	6.6	5.01	15.6	46.2	204	147.8	4.7	63[a]	6	2.3
	0P1	8.1	5.54	16.9	50.8	195	146.9	5.3[a]	61[a]	16	2.1
	00	6.8	5.38	16.7	49.3	202	148.9	4.7	63	11	2
	P1P1	5.6	5.06	15.8	46.7	191	147.8	5.1	68	8	2.5
	P2P1	8	5.36	16.7	48.5	251	148.5	4.5	70	11	2.3
30	P2GP1	6.2	5.06	15.8	46.3	192	148.6	4.9	69	282[a]	2.4
	HAP1	6.4	5.03	15.6	46.1	180	146.2	4.8	99	5	2.5
	0P1	8	5.43	16.7	49.5	174	147.3	5.1	82	9	1.8
	00	6.4	5.32	16.6	48.6	206	148.2	5	82	11	1.5[a]

Appendix 7. Continued

	P1P1	5.2	4.87	15.3	44.4	179	147.1	5.2	95	6	2.8
	P2P1	8.3	5.25	16.6	48.2	129	146.4	4.7	101	9	2.5
70	P2GP1	6.1	5.02	15.8	45.9	219	148.9	4.9	84	188[a]	2.6
	HAP1	6.5	4.97	15.4	45.9	202	146.3	4.9	95	6	2.1
	0P1	7.3	5.31	16.3	48.7	255	140.6	5	103	10	2
	00	6	5.08	16.5	47.1	241	146.7	5.2[a]	87	11	1.6[a]
						Subject REA					
	P1P1	2.9	5.7	16.2	47.5	251	144.9	4.2	121[a]	9	1.1[a]
	P2P1	4.3	5.43	15.5	45.7	290	145.1	4.1	142[a]	10	0.9[a]
−105	P2GP1	3.2[a]	5.63	16.2	47.4	292	143.8	4.1	126[a]	8	1.1[a]
	HAP1	3.2[a]	5.61	15.9	47.1	292	143.5	4.1	127[a]	9	0.9[a]
	0P1	4	5.54	15.9	46.5	268	144.2	4.4	125[a]	6	0.9[a]
	00	2.7[a]	5.55	15.8	46.8	278	145.4	4.2	152[a]	8	0.7[a]
	P1P1	2.5	5.42	15.5	45.3	284	142.3	4	132[a]	9	–
	P2P1	4.7	5.34	15.3	44.9	317	145.4	4	173[a]	7	–
−35	P2GP1	2.8[a]	5.52	16	46.5	305	143.1	4.2	143[a]	68	–
	HAP1	3.2[a]	5.35	15.1	44.9	283	143.8	4.5	97	8	–
	0P1	4.9	5.51	15.7	46.5	284	144.8	4.5	105	6	–
	00	2.4[a]	5.52	15.9	46.9	259	146.1	4.6	127[a]	7	–
	P1P1	2.5	5.43	15.6	45.1	261	142.6	3.88	130[a]	10	–
	P2P1	4.7	5.35	15.3	45.1	300	146.3	3.8	164[a]	15	–
−25	P2GP1	2.8[a]	5.51	15.9	46.7	300	145.6	4.1	141[a]	47	–
	HAP1	3.2[a]	5.3	15.4	44.5	272	143.4	4.28	98	9	–
	0P1	5	5.46	15.8	46.2	309	144.9	4.5	115	9	–
	00	2.4[a]	5.5	15.6	46.3	270	146.2	4..5	134[a]	9	–
	P1P1	Missing	Missing	Missing	Missing	Missing	142.1	4.2	103	10	1.0[a]
	P2P1	5.2	5.34	15.3	44.5	336	146.4	3.9	147[a]	12	1.1[a]
0	P2GP1	3[a]	5.5	15.7	46.2	300	143.4	4.3	168[a]	159	1.0[a]
	HAP1	3.3[a]	5.36	15.1	44.6	228	142.9	4.3	102	10	2.1
	0P1	5.4	5.41	15.7	45.7	319	144.8	4.7	113	8	0.7[a]
	00	2.6[a]	5.51	15.8	46.4	271	145.7	4.6	121[a]	9	0.7[a]

Appendix 7. Concluded

	P1P1	5	5.65	16.1	47.5	283	143.7	5	82	11	1.1[a]
	P2P1	6.8	5.45	15.9	45.7	341	148.2	4.4	110	20	1.1[a]
10	P2GP1	4.4	5.68	16.4	48	323	146.2	4.7	114	123	1.0[a]
	HAP1	3.9	5.52	15.8	46.5	306	147.2	4.9	97	11	2
	0P1	6.9	5.64	16.4	47.8	316	147.4	5.1	98	10	1.0[a]
	00	3.8	5.73	16.5	48.3	268	148.3	5.1	92	11	1.0[a]
	P1P1	Clotted	Clotted	Clotted	Clotted	Clotted	143.1	5.7[a]	94	14	2.1
	P2P1	7.6	5.61	16.1	47.3	345	148.6	4.8	108	19	1.0[a]
30	P2GP1	4.5	5.68	16.3	48.1	349	146.2	5	112	139	1.3[a]
	HAP1	4.3	5.48	15.6	46.1	315	145.3	5	102	10	1.8
	0P1	7.3	5.6	16.2	47.8	315	146.8	5.2	118[a]	9	0.9[a]
	00	3.6	5.73	16.5	48.6	288	148.8	5.4[a]	94	12	0.9[a]
	P1P1	3.3	5.61	15.9	47	353	141.7	5.2[a]	110	12	1.4[a]
	P2P1	8	5.48	16	45.8	382	147.9	5.2[a]	117	18	1.4[a]
70	P2GP1	4.4	5.61	16.2	47.3	362	146.1	5.4[a]	122[a]	130	1.7
	HAP1	4.2	5.36	15.6	45	309	146.6	5.3[a]	105	10	1.5[a]
	0P1	7.5	5.54	16.1	46.8	301	146.6	5.3[a]	106	8	1.2[a]
	00	3.6	5.66	16.2	47.8	277	148.1	5.7[a]	91	12	1.3[a]

[a]Abnormal value.
[b]Equivalent triglyceride concentration.

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J. E. Greenleaf, R. Looft-Wilson, J. L. Wisherd, P. P. Fung, A. C. Ertl, C. G. R. Jackson, P. R. Barnes, and L. G. Wong*

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Point of Contact: J. E. Greenleaf, Ames Research Center, MS 239-11, Moffett Field, CA 94035-1000; (415) 604-6604
*Shaklee U.S., Inc., San Francisco, California

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13. ABSTRACT *(Maximum 200 words)*

To test the hypothesis that drink composition is more important than drink osmolality (Osm) for maintaining and increasing plasma volume (PV) at rest and during exercise, six men (22–39 yr, 76.84 ± 16.19 kg, 2.99 ± 0.45 L/min $\dot{V}O_2$ peak) each underwent six treatments while sitting for 90 min ($\dot{V}O_2$ = 0.39 L/min) and then performed upright ergometer exercise for 70 min ($\dot{V}O_2$ = 2.08 ± 0.33 l/min, 70% ± 7% $\dot{V}O_2$ peak). Drink formulations (10 ml/kg body weight, $\overline{X}$ = 768 ml) for the sitting period were: P1 (55 mEq Na^+, 365 mOsm/kg H_2O), P2 (97.1 mEq Na^+, 791 mOsm/kg), P2G (113 mEq Na^+, 80 ml glycerol, 1,382 mOsm/kg), HyperAde (HA) (164 mEq Na^+, 253 mOsm/kg), and 01 and 02 (no drinking). The exercise drink (10 ml/kg, 768 ml) was P1 for all treatments except 02. Plasma volume at rest increased ($p < 0.05$) by 4.7% with P1 and by 7.9% with HA. Percent change in PV during exercise was +1% to +3% (NS) with HA; –6% to 0% (NS) with P1, P2, P2G, and 01; and –8% to –5% ($p < 0.05$) with 02. HyperAde, with the lowest osmolality (253 mOsm/kg), maintained PV at rest and during exercise, whereas the other drinks with lower Na^+ and higher osmolality (365 to 1,382 mOsm/kg) did not. But Performance 1 also increased PV at rest. Thus, drink composition may be more important than drink osmolality for increasing plasma volume at rest and for maintaining it during exercise.

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